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Obstetrics and Gynecology

Indications for Hysterectomy

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Hysterectomy has become one of the more commonplace operations. It may be one of the simplest operative procedures and it can be one of the most difficult. One can often judge by a careful examination preoperatively the difficulties which he will encounter at the operating table, but not always. Unexpected complications are encountered at operation which if not properly handled may increase the morbidity and mortality. Just before the war we performed one thousand successive hysterectomies with two deaths. Both were from pulmonary embolism and probably unavoidable. Nevertheless, in general the mortality from hysterectomy in the better hospitals is in the neighborhood of one percent. In addition there may be a more or less serious morbidity associated with the operation due to bladder, ureteral or bowel injuries. These complications have become more frequent in recent years due to the more universal performance of total hysterectomy. This is not to indicate that I am opposed to total hysterectomy. On the contrary, I believe the total operation is the better procedure and should be done in almost all instances. On our Service last year we performed total hysterectomy in 92 per cent of the cases. I hope the incidence will never become 100 per cent for, if it should, I would be forced to the conclusion that the staff had lost its surgical judgment. Our ward service is largely composed of colored women who have large fibroids which are often complicated by salpingitis and it is sometimes wiser to do the lesser operation because of technical difficulties in removing the cervix. When in the judgment of the operator the danger of removing the cervix exceeds the danger of cancer occurring in the cervical stump he had better do the subtotal operation.

Indications for Hysterectomy.

I have spoken of the difficulties sometimes encountered with hysterectomy not to discourage its performance when there is a sound indication, but to emphasize that it should not be undertaken unless there is a real indication.

I shall consider the specific indications for hysterectomy under the following headings:

1. The Indications for Vaginal Hysterectomy.
2. The Indications for Abdominal Hysterectomy for Benign Uterine Disease.
3. The Indications for Abdominal Hysterectomy with Operations for Adnexal Disease.
4. The Indications for Abdominal Hysterectomy for Malignant Uterine Disease.

Within the past several years vaginal hysterectomy has become increasingly popular with some operators. I think this is proper for it has definite advantages over the abdominal operation. In general, the postoperative discomfort is less and recuperation is more rapid. My indications for vaginal hysterectomy are few, but they occur with sufficient frequency to make it one of our more frequent operations.

Recurrent functional bleeding of sufficient severity to require definitive treatment is one of the more frequent indications. I emphasize **recurrent** bleeding because I believe curettage should always be done first to establish the diagnosis and for its possible curative effect. Under certain circumstances a second curettage may be advisable, but if the bleeding recurs with sufficient severity to make definitive treatment advisable, vaginal removal of the uterus is the operation of choice. I believe this is true whether the vagina is nulliparous or parous and even when there is no descensus.

The same operation is indicated when one is dealing with a small bleeding fibroid. If the uterus is more than twice its normal size, I believe it is better to remove it abdominally. I am cognizant of the fact that not infrequently a large free fibroid can be removed vaginally quite safely by morcellation in expert hands, but in average hands stunt surgery had best be avoided. The presence of a low midline scar indicating previous pelvic surgery should make one very cautious in selecting vaginal hysterectomy. It is not an absolute contraindication, but one should make quite certain by careful examination under anesthesia that the uterus is freely movable before attempting its removal vaginally. As a matter of fact it is an excellent plan when doing vaginal hysterectomy to have a table set up for laparotomy for use if difficulties are encountered vaginally requiring quick entrance of the abdomen. I have only once in my career had to make this change, but I believe in that instance it was life saving and hence worth the extra effort of preparedness.

Abdominal suspension of the retrodisplaced uterus is justly being done less and less frequently. I think that this trend is proper, but I happen to believe there is still an occasional indication for an intra-abdominal uterine suspension. Nevertheless, when a woman has really distressing symptoms from retrodisplacement with or without

first degree descensus and when she has in her opinion completed her family, I believe a vaginal hysterectomy with the indicated vaginal plastic repair is far superior to an intra-uterine suspension. Relief of symptoms is assured, and further trouble from a diseased uterus is impossible.

This brings up the problem of the more marked degrees of uterine prolapse in relation to vaginal hysterectomy. It may surprise some of you to hear me say that I do not regard it as the operation of choice when there is marked descensus. It does not fall within the domain of this paper to discuss in detail the technical advantages and disadvantages of the various types of operation for prolapse and I will simply state that after giving vaginal hysterectomy a trial in the treatment of the advanced degree of prolapse we found it wanting in too many instances after a long follow-up. A prolapsed vagina occurring after vaginal hysterectomy is extremely troublesome and very different to repair, preserving a functional vagina. Our present attitude is that each case of prolapse should be regarded individually and the operation done which is best suited to the case. On the whole the Spalding-Richardson operation has proved quite satisfactory and we have reserved the vaginal hysterectomy for those cases of marked prolapse in which benign disease of the uterus makes its removal desirable.

Before leaving the subject of vaginal hysterectomy I should like to say a word about the removal of the grossly normal uterus for severe dysmenorrhea. I have done the operation for this indication perhaps half a dozen times in my professional career and since the uteri are normal in size I have usually done the operation vaginally. In the woman approaching forty or beyond who has extreme menstrual pain and upon whom all conservative measures have been tried it does not seem sensible to me to perform presacral neurectomy with perhaps a 70 per cent chance of relief when a hysterectomy can offer her certain relief. I am assuming that such a patient will have had her children without relief of her menstrual pain or that she has given up the idea of childbearing. Some of my most grateful patients fall into this group, but they are few and, if one does the operation in properly selected cases, the number should remain small.

Now let us consider the indications for abdominal hysterectomy for benign uterine disease. Probably one of the greatest surgical sins of our generation is the unnecessary removal of the asymptomatic myomatous uterus. There are definite indications for removal of the fibroid uterus and one should be satisfied in his own mind that at least one of these symptoms is present before advising hysterectomy. These indications are as follows:

Excessive bleeding.

Discomfort or severe pain arising from the tumor.

Pressure on adjacent organs causing dysfunction such as the bladder, bowel or ureter.

Distortion of the abdomen.

Evidence suggesting malignant change.

Myomectomy is indicated at times because of the effect of the fibroid upon past, present or future pregnancies. Since this paper deals only with the indications for hysterectomy, a discussion of myomectomy is not pertinent.

Let us consider the above indications individually. Excessive bleeding at the time of menstruation may be and often is due to fibroids, but excessive bleeding from the fibroid uterus is not necessarily due to the fibroid. Other pathological lesions such as endometrial hyperplasia or endometrial polyps may be the cause and the fibroids may be incidental. After all, how frequently does one encounter even large fibroids with normal or even scanty menstruation? Hence, when menorrhagia has been present only a month or two, a curettage should be done instead of rushing into a hysterectomy. Curettage may accomplish several things: It will remove an endometrial polyp and stop the bleeding; it will remove the hyperplastic endometrium and temporarily or permanently relieve the patient. It will tell you whether the uterine cavity has become irregular due to the submucous position of the fibroid, which information may be of great value in deciding on hysterectomy in the future. By curettage, combined with cervical biopsy, a diagnosis of malignancy can be made or excluded. It is particularly important to carry out this simple diagnostic procedure when there has been intermenstrual bleeding.

Abdominal or pelvic discomfort or more severe pain is a frequent reason for operative interference. There are many causes for pain with fibroids; the pressure of a large but uncomplicated tumor on the pelvic nerves may give rise to pain, and tumors which are the site of extensive necrosis are also sometimes painful. In rare instances pedunculated fibroids twist and give rise to a clinical picture of acute abdominal pain much like one seen with a twisted ovarian tumor. In the author's experience fibroids twist on their pedicle more often postmenopausally and during pregnancy. The commonest cause for pain in our public ward experience is a complicating pelvic inflammatory disease, acute or chronic. A long-standing pelvic inflammatory residue that has been asymptomatic for months or years may become painful when the growing fibroid begins to stretch the pelvic adhesions. Circulatory disturbances and edema resulting from pressure of the tumor upon chronically infected tubes sometimes results in acute or sub-

acute painful exacerbations. Such pelvic inflammatory disease seldom responds well to palliation, and surgery is necessary soon or late. Dysmenorrhea, acquired in the fourth or the fifth decade, may be the outstanding symptom of the growth of fibroids. A common symptom complex resulting from fibroids at this time of life is menstrual pain, coupled with increased menstrual flow. Regardless of which one of the above conditions is responsible for the pain hysterectomy is indicated.

Evidence of pressure on near-by pelvic viscera is also an indication for treatment. The urinary bladder is the organ that suffers most often from such pressure, giving rise to frequency of urination. Although this symptom is common with large fibroids, it is remarkable how frequently one sees the pelvis filled with fibroids without any increased frequency of urination. The great ability of the bladder to function normally in spite of extreme distortion by pelvic tumors is truly remarkable. Occasionally, acute retention of urine results from a fibroid and necessitates surgical interference. We have seen this occur as the result of marked growth of the fibroid anteriorly, pressing the superior surface of the bladder against the internal sphincter region. More frequently a tumor of about the size of a 3-months' pregnancy incarcerated in the cul-de-sac pushes the cervix downward and forward and obstructs the flow from the urethra. We have also observed a large pedunculated submucous tumor, filling and distending the vagina, pressing on the urethra and causing retention. As stated previously, the pressure of the tumor upon the ureters at the pelvic brim, with resultant kidney damage, may indicate the necessity of operative treatment.

The bowel is less apt to show symptoms from pressure than the bladder, but constipation can be caused and aggravated by such pressure; more frequently one is astounded by the relatively normal function of the bowel in the presence of large fibroids, that almost completely fill the pelvis. Very rarely we have seen acute intestinal obstruction occur from pressure of a large fibroid on the bowel.

Often a large fibroid uterus will give no other symptoms except abdominal distention. No woman should be required to appear as though perpetually pregnant or to have a permanently distorted figure and I believe this constitutes a reason for hysterectomy even without other symptoms.

When a woman is told that she has a fibroid uterus and is advised that it may be safely kept under observation the natural question is, "What about malignancy?" The chance of fibroids undergoing malignant change is slight. Evans in a large series of fibroids at the Mayo Clinic found sarcomatous change in only 0.7 per cent. Actually the percentage of malignancy in fibroids is much less than that, for this percentage was based on the fibroids removed for proper indications. If the percentage of malignancy could have been based on all existing fibroids (which is obviously impossible), it would have been much smaller. Certain it is that the risk of malignant change is no greater than the risk of surgery in most instances so the possibility of its occurrence does not constitute a legitimate reason for removal. On the other hand, when there is reason to suspect malignant change because of rapid growth, or any growth after the menopause, hysterectomy is indicated. Rarely, a benign myoma grows after the menopause, but this is so rare that it is only safe to assume that proven postmenopausal growth indicates sarcomatous change and immediate hysterectomy.

What are the indications for hysterectomy when surgery is done for adnexal disease? This question should be answered in three parts depending upon whether we are dealing with salpingitis, endometriosis or ovarian tumors.

It is recognized generally today that surgery is usually done for the residue of salpingitis and the resultant pelvic pain, rather than for the elimination of the infection. Before the advent of the sulfas or antibiotics it was recognized that acute and subacute salpingitis would usually subside with rest, local heat and the passage of time. Only the local complications, such as pelvic abscess, require drainage. But rarely a thick walled tubo-ovarian abscess with thorough antibiotic treatment persists and requires removal to eliminate the infection. Nevertheless, most surgery today is and should be done for persistent abdominal and pelvic pain due to adhesions resulting from the burned out infection. Since Neisserian tubal infection is practically always bilateral, a double salpingectomy is required, sometimes with the removal of one or both ovaries. With postabortal infection bilaterality is not as often the rule and if surgery is required a normal tube may at times be spared. When I took over the ward service at Hopkins I was immediately struck with the number of patients who required further surgery for uterine disease upon whom bilateral salpingectomy had been done several years before. In the sterile woman the uterus can be nothing but a liability and in recent years it is almost an invariable rule to perform hysterectomy when both tubes are removed. This probability is discussed with the patient pre-operatively and if the patient is adamant in her desire to have her uterus spared we may yield to her desire. When the tubal disease is tuberculous, hysterectomy is still more important because the uterus frequently is involved in the tuberculous disease.

When the severity of the symptoms requires laparotomy for endometriosis one may have the choice of conservatism with preservation of the

child bearing function, radical surgery and semi-radical surgery. By the last procedure we mean hysterectomy but preservation of at least a portion of one ovary. Naturally the younger the woman and the greater her desire for pregnancy the harder one attempts to be conservative. Our results with conservative surgery in these young women have been most gratifying. In the woman past forty with extensive involvement of both ovaries with endometriosis, hysterectomy and double adnexectomy are usually the procedures of choice. But there is a group of relatively young women with endometriosis in whom child bearing is past or impossible in whom we have been able to avoid complete castration but in whom the removal of the uterus should be done to eliminate severe menstrual pain. Although a small percentage of these women may ultimately require radical surgery the majority of them are kept comfortable by this semi-radical procedure.

There is no place in all surgery when a knowledge of gross pathology is more important than when dealing with ovarian neoplasms. When dealing with unilateral benign tumors in relatively young women it is obvious that the uterus and opposite ovary should be spared. With benign unilateral tumors in the middle aged woman approaching her menopause it is often wiser to remove the opposite adnexa and uterus. With malignant bilateral ovarian cancer there is, of course, no choice but to perform hysterectomy and double adnexectomy. But what of the unilateral, well encapsulated malignant ovarian tumor? Radical surgery is the back bone of the treatment of almost all malignant ovarian neoplasms. There is a temptation at times to simply remove the affected ovary and tube but the lymphatic connections between the uterus and opposite adnexa are so extensive that radical surgery should be done. Even when the opposite ovary appears normal, microscopic cancer is present in a high percentage of the cases. In fact, if a mistake has been made and a unilateral ovarian neoplasm is removed without removal of the uterus, only to find the tumor malignant on microscopic section we believe a second operation should be done without delay for the removal of the uterus and opposite adnexa. There is only one exception which we make to the rule of radical surgery for malignant ovarian tumors. In very young individuals one occasionally encounters a feminizing or masculinizing well encapsulated tumor. Although these tumors are malignant they are usually of low grade malignancy and a calculated risk of conservative surgery may be taken.

The question of hysterectomy for carcinoma of the corpus uteri is not controversial. All gynecologists in the United States and Canada are agreed that total hysterectomy and bilateral salpingo-oophorectomy should be done for endometrial cancer. All are not agreed on the advantage of preoperative irradiation. In our clinic we believe it is advantageous on the basis of our own experience and an unbiased review of the literature. Recurrences in the vaginal vault appear to be fewer if preoperative intracavitary irradiation is done.

The question of hysterectomy for cervical cancer is very controversial. Opinion is varied not only regarding surgery versus irradiation but there are some who believe a combination of the two should be used. I can only give you our views and attempt to give our reasons for holding them. In selecting therapy for cervical cancer today one should make as exact an estimate as possible as to the extent of the growth. Almost everyone is agreed that stages 3 and 4 of cervical cancer should be treated by irradiation. The results are poor but the only surgical attack possible is by exenteration which does not improve the salvage, has a terrific mortality and still greater morbidity. At the other extreme is Stage O, the preinvasive growths. It is our custom to treat them by surgery. The operation which we do for this stage of the disease is a Modified Wertheim hysterectomy. By this we mean that we remove two or three centimeters of parametrium and a good vaginal cuff but we do not do a pelvic lymphadenectomy. In our experiment with therapy with this stage of cervical cancer we decided on this type of operation and now have operated upon over two hundred cases within the last 12 years and all are living and well. In addition, since so many of these occur in young women we frequently save an ovary and thus prevent an early menopause. That this is a safe practice is shown by the fact that all of these patients are also well. For these reasons we have continued with this treatment of Stage O.

Most gynecologists concede that Stage 2 of the disease, in which there is extension to the upper vagina and to a slight degree to the parametrium, should be irradiated. The real controversy is concerning Stage 1, when the entire lesion is confined to the cervix. It has been our custom to treat these women with a full course of irradiation. Our 5 year salvage has reached 85 per cent. At the Massachusetts General Hospital, Dr. Meigs has treated this group of patients by the radical hysterectomy, adnexectomy and pelvic lymphadenectomy. His salvage is practically identical to ours, but this major surgery is much more an ordeal for the patient than is the application of radium, and urinary tract complications are more frequent among the surgical cases. In our clinic we reserve this operation for the Stage 1 and occasional early Stage 2 cases which do not respond to irradiation or have local recurrence in the cervix. In deciding the question of irradiation versus surgery one should always bear in mind that the results of Meigs are those of an expert

technician in pelvic surgery who has had long and constant experience with this operation. The average or even good pelvic surgeon who might be called upon to perform the operation only occasionally had better decide on irradiation even in what appear to be favorable Stage 1 cases.

Bacteriology

Adenoidal-Pharyngeal-Conjunctival Virus Diseases

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In 1953 Huebner et al reported the isolation from human adenoids of an agent with the general characteristics of a virus[1]. At that time, adenoids removed from young children were being examined in the laboratory as a potential source of tissue culture for virus isolation; it was observed that some of these cells began to degenerate. Material from the degenerated adenoidal cells was subcultured to other types of tissue cultures ordinarily used for virus isolation. When a similar degeneration of these tissue culture cells also occurred, it was postulated that a cytopathogenic agent had been isolated. This agent was demonstrated to be filterable, unable to multiply on ordinary bacteriological media, and was invisible with the ordinary microscope. It was therefore considered to be probably a virus or a rickettsiae and was designated as the "adenoid degeneration" agent or the "A. D." agent.

Subsequent investigations[2] carried out with this agent indicated that it was capable of producing unique cytopathogenic effects on several different kinds of tissue culture, but was non pathogenic to laboratory animals. It was resistant to antibiotics and to ether, but fairly sensitive to heat. Six different serologic types (I to VI) of the agent and later a seventh (VII) were identified after observing that sera from some patients neutralized only some strains of the virus. As some of these viruses were isolated from conjunctiva the designation of the group was changed from "adenoid degeneration" agent to "adenoidal-pharyngeal-conjunctival" agents or "A. P. C." agents.

Hilleman[3] in 1954, reported a new agent recovered from patients with acute respiratory illness who showed the principal evidence of disease in the chest. He designated this agent as the "acute respiratory disease" agent or "A. R. D." agent, or more specifically R1-67 agent. On comparison of the A. R. D. agent with Huebner's A. P. C. agents it was noted that the A. R. D. agent had the same general characteristics as the A. P. C. agents and overlapped in some neutralization tests. The A. R. D. agent was then included with the A. P. C. viruses as Type IV A. P. C. virus.

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The isolation of a microorganism from a patient does not by itself establish as fact that the disease from which the patient is suffering is produced by that microorganism, although additional support may be given to such a conclusion if during the course of the patient's illness a rising antibody titer against the isolated agent can be demonstrated. The final diagnosis, however, must be based on the fact that the symptoms and nature of the disease in the patient are similar to the infection usually produced following invasion by the microorganism. The nature of the diseases produced by many different microorganisms has now been well established by clinical observation and animal experimentation. Since the A. P. C. agents do not produce disease in animals, it has been necessary to rely on clinical observations alone in an endeavor to establish the nature of the diseases in patients from whom the A. P. C. agents have been isolated by means of tissue culture. As the first isolation of this virus was made only two years ago, the clinical syndromes associated with the isolation of this group of agents have not been entirely worked out. Some observations however have been made.

Types I, II, V and VI are generally regarded as commensals, recoverable from tonsils and adenoids usually showing no evidence of clinically active inflammation. The recovery of these types of filterable agents may therefore be comparable to the recovery of **Streptococcus viridans** and of Diphtheroids from tonsils.

Types III, IV and recently Type VII have been recovered from throat swabs, from stool specimens or from conjunctival swabs of patients with acute respiratory infections with symptoms persisting for an average of five to six days. Although both children and adults may be infected, the disease is probably more commonly seen in children and is of only three to four days duration. Symptoms superficially resemble those of the common cold, including sore throat and nasal discharge, fever and chills; some patients complain of cough and anorexia, and children may vomit. A fairly high percentage of patients complain primarily of conjunctivitis; a few of tenderness in the right upper quadrant, and in some patients the distress is centered in the chest with coughing the principal complaint.

Examination of these patients usually shows a mild pharyngitis and tonsillitis with a congestion

of nasal mucosa. Conjunctivitis is frequently unilateral and troublesome. The patient's temperature may reach 104°F and an elevation of temperature may persist from five to six days. Liver tenderness may be observed in a few patients. Patients complaining of soreness of the chest and with severe coughing may show changes on the chest roentgenogram similar to those of an atypical pneumonia; the cold agglutinin test in this group of A. P. C. viruses being negative.

Since the A. P. C. virus infections are probably widespread, these agents may be isolated from patients suffering from other types of diseases or infections. It is therefore necessary to hesitate before making a diagnosis of A. P. C. infection based on isolation of the virus plus the relatively limited clinical observations of these diseases. More experience will be required before the diseases associated with this group of viruses can be entirely defined.

Huebner et al have investigated the prevalence of previous infections with this group of viruses, by studying the level of serum antibodies in different population groups. It was noted that the antibody content of different sera varied with different age groups and with different types of A. P. C. viruses. This investigation revealed that in the majority of infants from six to eleven months of age, antibodies were present against at least one type of A. P. C. virus, but with an increase in age a corresponding increase in antibodies occurred so that the majority of adults showed antibodies to three or more types of A. P. C. viruses.

Some of the material submitted to the Manitoba Virus Laboratory for study has been obtained from patients with acute respiratory and eye infections. These specimens have included throat swabs, throat washings, samples of feces, and conjunctival swabs and scrapings. Specimens for examination are first treated to remove all microorganisms other than virus and are then inoculated on tissue culture (HeLa cells and monkey kidney cells); these cultures are observed daily for a cytopathogenic effect. Any of the viruses isolated that may belong to the A. P. C. group are identified with type specific rabbit antisera.

Blood samples from patients in the acute and convalescent stages of disease are examined by the complement fixation test to demonstrate a rise in antibody titer against the A. P. C. group of viruses. The complement fixation test, however, does not identify the type of the infecting virus but only that it is an A. P. C. virus infection; to determine the specific type of antibody present, blood samples may be examined by neutralization tests on tissue culture.

A. P. C. virus has been isolated from specimens submitted from patients in this province. Six of the isolations were made from material obtained from patients showing primarily an eye infection. Five isolations were made from material of patients suffering primarily from a pharyngitis and tonsillitis, while the remaining four isolations were made from material taken from patients showing chest symptoms. Seven of the viruses isolated have been identified specifically as Type III A. P. C. virus.

In addition to the patients from whom A. P. C. virus are successfully isolated we have been able to demonstrate an increase in antibody titer against the virus in six other patients; four of these patients complained primarily of a tonsillitis and pharyngitis, and two of a moderately severe chest infection with roentgen changes in the pulmonary parenchyma.

At present a survey is being carried out in the Manitoba Virus Laboratory to determine the distribution of antibodies in the sera of children of six months to six years of age against Type III A. P. C. virus, which appears to be prevalent in this area. Specimens for this examination are submitted by the Children's Hospital in Winnipeg.

Material to be submitted for examination of A. P. C. virus infections includes the following:

(1) Swabs of pharynx and tonsils.

(2) Swabs of conjunctival sac.
The number of positive cultures are increased if the patient can be sent directly to the laboratory.

(3) Two specimens of feces collected early in the course of the disease.

(4) Two blood specimens, the first one at the onset of the disease and second one two weeks later. These are collected in a Keidel tube and allowed to clot; they should be submitted as soon as possible after collection.

Samples may be submitted to the:
Virus Research Laboratory,
Room 126,
Medical College Buildings,
Bannatyne Ave. and Emily St.,
Winnipeg, Manitoba.

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Surgery

Operating Room Cholangiography

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Operating-room cholangiography has commanded a varying degree of enthusiasm among surgeons since the method was first suggested by M. S. Cotte in his paper before "La Société Nationale de Chirirgie" in Paris in 1929[3]. The paper illustrated the use of the routine post-operative T-tube cholangiogram as a method of checking on the thoroughness with which the common-duct has been explored before the removal of the T-tube; as an aside, he mentioned operative cholangiography as a more desirable check though more difficult of realisation. P. L. Mirizzi of Cordova, Argentine, wrote a series of papers on the method and advantages of operative cholangiography and published the results of ten years utilization of the method in 1939. Since the pioneer efforts of this author, the technique of operative cholangiography has undergone many refinements and is now known to be as reliable as the well established post-operative cholangiogram.

The term operative cholangiogram embraces two procedures; namely, the "diagnostic" operative cholangiogram which is performed before the common duct is opened usually through the cystic duct using a polyethylene catheter; and the "control" operative cholangiogram which is performed through the T-tube inserted into the common-duct at the completion of an exploratory choledochostomy.

Advantages of Operative Cholangiography:

1. As a supplementary method of examining the biliary tree containing biliary calculi.

Frank Glenn has stated that in all patients with stones in the gall-bladder, one in ten will have a stone in the common-duct[6]. By the process of obstruction, many of these stones will produce recognizable changes in the appearance of the common-duct. Others will produce questionable changes. In the latter instance, cholangiography, before opening the common-duct, can serve as a screening test for symptomless stones.

The use of "diagnostic" operating-room cholangiography as a method for exploring the biliary tree for calculi in search for a specific indication for choledochostomy has been studied by several authors; some, such as Hughes and Kernett[8], Waltman Walters[13], find the method disappointing; others have found the procedure worthwhile. Mixter, Hermanson and Segel[9] in the analysis of 107 cholangiograms on common-ducts, which had one of more generally accepted clinical indications for choledochostomy, which were not opened on the basis of normal operative cholangiograms, found that 7 had biliary colic on follow-up study (4.5%). On the basis of this study they concluded that it is difficult to demonstrate small stones or detritus by this technique in the absence of obstruction. They decided that a normal cholangiogram is a trustworthy indication for avoiding choledochostomy except when the gall-bladder contains small stones or the common-duct contains muddy bile and needs drainage because of cholangitis.

These same authors analyzed 146 "control" cholangiograms following choledochostomy for stones, prior to closing the abdomen. In 13% of cases the cholangiogram revealed overlooked stones. In 4.9% of cases stones were subsequently found at secondary operations or at post-mortem study in spite of the presumed operative clearance of ducts and negative check cholangiograms. They concluded, that though not foolproof, the method gives additional assurance that no stone has been left behind.

2. An indispensable aid in secondary explorations of the biliary tree.

Taken during the early part of the dissection an operative "diagnostic" cholangiogram greatly facilitates the exploratory procedure, avoiding needless and possible harmful dissection. In the case of a completely transected common-duct, an outline of the proximal ducts by cholangiography would give an idea of the length of common-duct from the point of section to the juncture of the hepatic ducts available for anastomosis. Where a stricture exists a cholangiogram will give exact information as to the site and length of the stricture. It may demonstrate the presence of multiple lesions such as calculi and stricture, which may be missed by the instrumental method of exploration.

3. An indispensable aid in exploring the distal end of the common-duct.

The demonstration of a common channel between the common duct and the pancreatic duct by the reflux of dye into the pancreatic duct plus evidence of stenosis or spasm at the papilla, in a patient suffering from recurrent pancreatitis may be an indication for a sphincterotomy as advocated by Mahorner. A stone in the distal end of the common-duct is in a position difficult to explore by palpation and easily demonstrated by cholangiography. A constriction of the distal common-duct, due to pancreatitis or pancreatic carcinoma is also easily demonstrated by this method.

4. An aid in surgery for jaundice[7].

Hepatitis or severe cirrhosis may be suspected to be the primary cause of the jaundice, but the medical attendants may be unwilling to deny the

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patient the benefit of surgical relief of a possible obstruction. Such a patient is often a poor candidate for surgery and in addition, if he has cirrhosis, a large liver and venous collaterals will increase technical difficulties. A normal cholangiogram would prove hepatic origin of the jaundice and avoid a needless exploration. Where obstruction is the cause of the jaundice, a cholangiogram would differentiate the cause of obstruction of the common-duct, be it due to stricture, calculus, tumor or fibrosis of the sphincter of Oddi.

5. An aid in surgery for biliary obstruction in Infancy.

Surgical exploration must be anatomically complete and often involves a difficult and time consuming dissection which with the aid of a cholangiogram could be rendered as atraumatic as possible. The differential diagnosis of prolonged jaundice in infancy can be difficult and may require operative exploration for determination. Ladd and Gross found only 20% of cases of atresia of the extra-biliary tree to be operable.

Ericsson and Rudhe[5] reported on the exploration of eleven cases of prolonged jaundice in infancy and found three to be due to cirrhosis without common-duct atresia as determined by liver biopsy and operative cholangiography. They found that cholangiography was a great aid in dissection and proper surgical treatment. Where the cholangiogram was normal the necessity of further surgical exploration was obviated. Where obstruction existed, the cholangiogram demonstrated the site of obstruction, and the size and availability of ducts for anastomosis to the intestinal tract. Cholangiography gave information of the state and the caliber of the intrahepatic ducts which would be impossible to demonstrate otherwise.

6. To demonstrate unusual lesions which might be difficult or impossible to find by the ordinary methods of inspection, palpation or exploration.

Examples of these lesions would include early carcinoma of the head of the pancreas, intrahepatic carcinoma of the ducts, strictures of intrahepatic portions of hepatic ducts and extrinsic obstructions.

Technique of Operative Cholangiography.

Diagnostic operative cholangiography is performed by injection of a 35% solution of Diodrast through a polyethylene tube into the gall-bladder, cystic or common-duct depending on the indication; the "control" cholangiogram is performed by injection of this solution through a T-tube into the common-duct.

Recently the use of a flanged polyethylene tube, as described by Block and Orloff, has been adopted at the St. Boniface Hospital. There are several advantages in the use of this tube. The transparency of the tube permits easy elimination of air from the injection by filling the tube with aspirated bile before the injection of the radio-opaque solution is begun. The long length of the tube, from one to two feet, permits the operator to remain well out of the X-ray field. The flange placed at a point two centimeters from the end of the tube, permits control over the amount of tube inserted into the ducts and also prevents an inadvertent withdrawal of the tube out of the ducts during cholangiography.

Some form of transverse incision is favored to permit the polyethylene tube to lie lateral to the biliary tree; for if the tube enters the abdomen through a mid-line incision it would overlie the biliary ducts and produce an imperfect film.

In order to get satisfactory films the following rules are observed:

1. The operative field is cleared of instruments which may overlie the extrabiliary tree and abdominal packs which may distort the ducts by external pressure.

2. At least two films are taken: one after the injection of 5 cc. of dye, a second after 15 to 30 cc. of dye, depending on the capacity of the biliary tree. The purpose of the first film is to demonstrate small calculi which may be obscured by a large quantity of dye.

3. Previous to the injection the operator must be sure his syringe and tubing are free of air by aspiration of bile; for air bubbles injected into the common-duct would produce filling defects indistinguishable from calculi.

4. During exposure of the film the anaesthetist renders the patient apneic either by previous hyperventilation or by the injection of a muscle relaxant such as succinyl choline.

5. The injection is made with a syringe using a minimum of force to prevent overdistention of the common-duct which can cause a spasm of the sphincter of Oddi.

It has been repeatedly emphasized by authors on this subject that not only interest but absolute co-operation must be maintained among the surgeon, the anaesthetist and the radiologist during the procedure; the slightest miscalculation on the part of any one of the team will nullify the entire examination or necessitate its repetition. Apart from other arguments, in order to acquire proficiency in operating room cholangiography some authors[10, 11] urge the routine use of the procedure in all operations on the biliary tree. This would be difficult to realize and of questionable value in our institution. In view of the relatively high incidence of missed common-duct stones and the very limited practical value of post-operative cholangiography, a control operative cholangiogram as originally advocated by Cotte, could be advantageously performed at the conclusion of a choledochostomy.

The Interpretation of Operative Cholangiograms.

The following are the common defects produced by pathological conditions of the biliary tree. A

small round filling defect in the lumen of the common-duct is produced by small calculi. A convex blunting of the ampullary end of the common-duct indicates the presence of an impacted calculus. Dilatation is a sign of organic obstruction. Narrowing of the common-duct with proximal dilatation may be seen in benign strictures, pancreatitis and carcinoma of the pancreas. A funnel shaped deformity of the distal end of a dilated common-duct is usually produced by a carcinoma of the head of the pancreas.

Sources of Error in Operative Cholangiography.

1. Diaphragmatic motion during exposure of the film will produce a blurred picture.

2 Air in the biliary tree will produce filling defects indistinguishable from calculi.

3. Duodenal injection by the insertion of an excessive length of polyethylene tube through the ampulla. This is avoided by the use of the flanged tube; for the flange is drawn up against the purse-string suture surrounding the stab wound through which the tube is inserted into the biliary tree.

4. Incomplete filling due to failure to estimate the necessary quantity of dye needed, external pressure on ducts by laparotomy pads, or a distended stomach, or too rapid emptying of the dye into the duodenum. Cholangiography in infants may suffer from incomplete filling due to too rapid emptying of the dye into the duodenum; the intrahepatic ducts may be more satisfactorily filled by compression of the lower end of the common-duct during injection[5].

5. The interposition of forceps, towel clips, and coagulation unit grounding plates may interfere with proper exposure of the film.

6. Periductal leaking of the dye may occur if the needle or straight ureteral catheter used for the injection slips out of the biliary duct during injection. This is avoided by the use of the cuffed polyethylene tube.

7. Errors in interpretation.

At the St. Boniface Hospital, operating-room cholangiograms are taken with the use of a slotted auxiliary full length table top which is placed on top of the ordinary operating table and covered with a sponge rubber plastic cushion, as described by Young and Scanlon[13]. The films are placed in grid front cassettes and wound into position by an endless chain operated from the foot end of the table. The X-rays are taken by the use of a portable fully rectified 100 Kv X-ray machine. As a rule a preliminary plate is taken before the operation as a check on the exposure time and type of exposure necessary for a proper film.

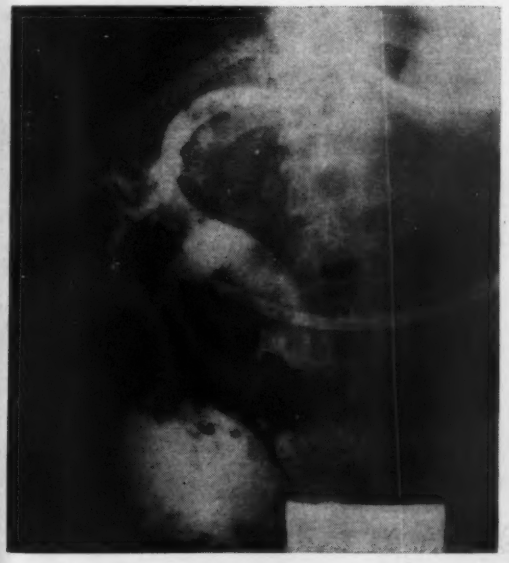

Figure 1

This is the "diagnostic" operative cholangiogram performed through a cuffed polyethylene tube inserted into the gall-bladder. Due to difficulty in reproduction the fine polyethylene tube is not visible in this illustration. The distal end of the gall-bladder is not filled due to the presence of impacted stones. The common and right hepatic duct are dilated and contain numerous calculi, the largest of which is at the distal end of the common bile duct, represented by intra-luminal filling defects. A Levine tube is seen to pass through the Stomach and into the first part of the duodenum.

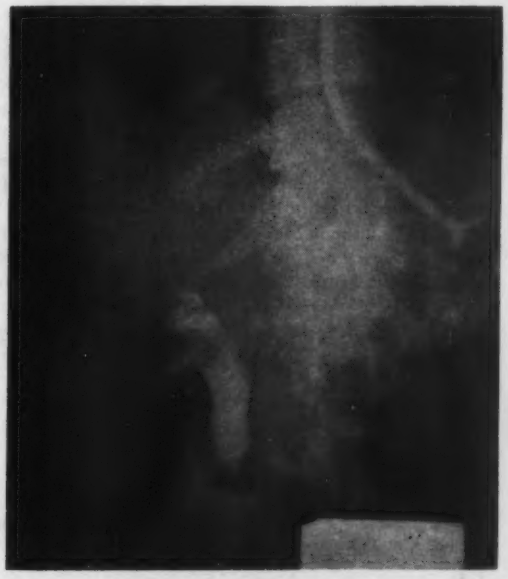

Figure 2

This is the "control" operative cholangiogram on the reported case. The radio-opaque dye was injected through the T-tube which enters the common duct from the viewer's left. The incomplete gall-bladder was removed flush with the thick-walled common duct and the defect closed transversely as seen at the upper end of the common duct. The lumen of the common duct can be seen to be free of calculi. Though not foolproof, a negative "control" cholangiogram gives reassurance of complete clearing of the common bile duct.

The following is a report of a case in which the use of an operating room cholangiogram proved to be of value.

A 74 year old white male was admitted as a Staff patient to the St. Boniface Hospital on August 21, 1955 with jaundice, pain in the right upper quadrant of the abdomen, and chills and fever of 3 weeks duration. The history of the present illness began with a severe right upper quadrant pain together with chills and fever followed 2 - 3 days later by jaundice. The jaundice is said to have become progressively worse. Previous to the present illness the patient admitted having an episode of epigastric pain a year before admission. Physical examination revealed a jaundice, emaciated male who had a tender liver palpable two fingerbreadths below the right costal margin. On admission, a urinalysis was positive for bile. The R.B.C. was 2.9 million with 9 grams % of hemoglobin. The total serum bilirubin was 2.2 mgms.%. The cephalin cholesterol flocculation was 3 plus. The prothrombin time 90% of normal. The serum alkaline phosphotase was 118 Bodanski units.

A pre-operative diagnosis of obstructive jaundice with cholangitis was made and an operation performed through a subcostal incision on August 26. During the dissection of the gall-bladder a fistulus tract was divided between the bladder and the first portion of the duodenum. There was difficulty in finding the cystic duct and as the dissection proceded along what appeared to be a cleavage plane, a hole was made inadvertently in the gall - bladder. It was then decided to do a "diagnostic" cholangiogram by inserting the flanged polyethylene tube through the hole in the gall-bladder. The gall-bladder which contained calculi was found to be small and directly communicating with greatly dilated common - duct without the intermediary of a cystic duct. The common-duct contained numerous calculi and appeared to empty into the third portion of the duodenum. After removing the calculi from the common-duct and amputating the gall-bladder, a T-tube was inserted through the choledochostomy opening and a "control" cholangiogram to check for remaining stones was performed

The use of the diagnostic cholangiogram to demonstrate the anomalous anatomy of the bile ducts greatly facilitated dissection.

Conclusions.

Though the routine diagnostic cholangiogram for all operations on the biliary tree is neither necessary nor feasible except in research institutions, its usefulness as an adjunct in the exploration of the common duct as a planned procedure is unquestionable and should be considered routinely. Operative cholangiography in the exploration of the main bile ducts may some day be recognized to be as valuable a diagnostic tool as the barium meal X-ray examination has proved to be of value in gastro-intestinal surgery.

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Public Health

Epidemiology of Infectious Hepatitis

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Our knowledge of infectious hepatitis, at the moment, is very limited. We are in the same stage now in the investigation of the disease as William Budd was over one hundred years ago when he was studying typhoid. However, his recommendations on the control of typhoid fever are carried out today, even though he made those recommendations long before the organism was discovered. Epidemiology, therefore, is a vital science in the study of infectious hepatitis today because, although we believe that the cause of the latter is a virus, its nature has not yet been fully understood. As far as we know, it can only be transmitted from man to man, and, therefore, all animal experiments are, at present, of no avail.

The attack made by this virus on the liver is similar in its destructiveness to that seen by another virus causing the disease syndrome known as homologous serum jaundice. Recovery of the liver is the rule, although some cases are known to die.

In 1953, ninety-four, (94), people died in Canada of this disease[1]. Because the full number of cases are not known owing to incomplete reporting, the case fatality rate cannot yet be ascertained.

Seasonal Incidence.

The disease is, we believe, one that appears predominately during the winter months, but cases are reported during every week of the year. In Canada during 1953 the number of cases reported totalled 2,755[2]. There was a peak incidence during the period between January 25th and February 21st, followed by a steady decline reaching its lowest ebb between August 9th and September 5th, but rising very steeply towards another peak period between 29th November and December 26th. It seems therefore that Canada experienced two major episodes of the disease during the year, one well before Christmas and the other well after the New Year. The United States Public Health Service has been receiving reports of infectious hepatitis cases during 1953 and 1954, and from graphs portrayed by them in the present issue of "Public Health Reports"[3] it appears that they, too, experienced these twin peaks, one in December and the other at the end of February. Barron[4] reported on an epidemic among recruits and young soldiers in the Kingston, Ontario area during 1949 and 1950. He, too, observed the occurrence of two peak episodes in the epidemic. In his study he claims that the second peak which was unexpected, fell rapidly after the institution of a water sterilization program.

The fact that these peaks are so uniform in the reports coming in, suggests strongly that there is a factor occurring at this time of year responsible for such increases. I would not be in a position to offer any explanation for this seasonal occurrence, but with a knowledge that the incubation period for this disease is approximately 30 days the period of greatest infectivity would coincide with the holiday festivities common at Thanksgiving and Christmas. It is at these times that people associate with each other and are at each others' homes. Conversely, the period of least infectivity which would occur 30 days prior to the period of lowest incidence in reported cases would occur during the last half of July and beginning of August when outdoor life is at its height. As one studies this disease more and more in detail, one feels that, perhaps, this might offer a possible explanation of the mode of spread.

Income Status.

This disease is believed to be spread in places where low standards of personal hygiene exist. The study of sixty epidemics of infectious hepatitis received by the United States Public Health Service involving just less than 3,000 cases indicates that the low socio-economic group was chiefly involved[3]. Lilienfeld et al, observed in an outbreak amongst 109 cases of jaundice at Baltimore in 1951 that there was no influence from income status[5]; and Spooner reporting on the incidence amongst certain 8th Army Regiments showed that Army officers were more prone to the disease than those in the ranks by 4.7: 1[6]. It is therefore difficult to earmark any particular type of person, but those with poor personal hygiene seem either more likely to contact the disease or to spread it to others, however careful the latter may be in their own hygiene. This last statement depends upon the acceptance of the fact that transmission is possible through indirect contact.

Age Group Most Commonly Attacked.

Infectious hepatitis is essentially a disease of young people, although all ages are attacked. The very fact that Gamma Globulin is an effective preventive, as we shall see later, makes it appear reasonable that the majority of people over the age of thirty are immune. Efforts have been made in various studies to pin down a particular age group for the highest incidence of this disease. Most of these studies, however, have been based on cases of jaundice, and we now know that jaundice is only an occasional symptom of the disease. It is safe to say that infectious hepatitis is most commonly experienced under the age of thirty or less.

Severity of Disease.

The severity of the disease appears to be dependent upon the age of the person[8], the concentration of virus present, and the degree of attenuation of the organism[7]. As far as age is concerned, (Table I), the Canadian death rates of this disease in 1953 rose noticeably after the age of 55, showing only minor differences before that age.

Table 1

Infectious Hepatitis: Canada 1953
Age Specific Death Rate

Age	Population in 1000's	Deaths from Infectious Hepatitis	Death Rates per 100,000 Population
0 - 4	1830.7	12	0.7
5 - 9	1536.5	5	0.3
10 - 14	1227.7	7	0.6
15 - 19	1060.3	—	—
20 - 24	1099.2	4	0.4
25 - 29	1169.1	5	0.4
30 - 34	1096.0	1	0.1
35 - 39	1045.7	6	0.6
40 - 44	930.8	8	0.9
45 - 49	792.9	3	0.4
50 - 54	690.3	5	0.7
55 - 59	594.8	8	1.3
60 - 64	512.8	8	1.6
65 - 69	444.2	6	1.3
70 - 74	337.9	3	0.9
75 - 79	207.7	5	2.4
80 - 84	104.5	5	4.8
85.	54.9	3	5.5

The death rate in the 0-4 age group was 0.7 per 100,000 children; but for those under the age of One, (Table II), the rate is higher at 1.7. The general rule, however, is one of complete recovery, although we must not forget that infectious hepatitis comes high in the death causes from communicable disease at all ages.

Table 2

Infectious Hepatitis: Canada, 1953
Death Rate for Under One

Estimated Live Births Mid-year 1952 to Mid-year 1953	Infectious Hepatitis Deaths under One 1953	Death Rate per 100,000
409,675	7	1.7

Symptoms.

Barron[4] in his investigation of an epidemic amongst Army installations in the Kingston area of Ontario found among 260 cases the following symptoms: abdominal distress, 82%, malaise, 74%, anorexia, 74%, nausea, 43%, vomiting, 38%, fever on admission, 29%, jaundice under 5%. 89% of these cases were between recruiting age and thirty.

Stokes[8], however, reporting on an endemic hepatitis in a Chicago Orphanage noticed a particular selection by the disease of nurses-in-training and medical students rather than the infants; but he later proved that these infants were also infected. Thus, at this age it seems that the disease can be almost asymptomatic.

The diagnosis of this disease cannot be based, therefore, on the occurrence of jaundice, although this symptom remains our guiding factor in the pin-pointing of cases of infectious hepatitis in a community.

Diagnosis.

Of the 260 cases investigated by Barron[4], jaundice occurred in only 15, (4.8%). Diagnosis was based upon the finding of bile in the urine—253 cases, (97%). Liver dysfunction—14 cases, (5.4%), and a rise above normal of the Icterus Index in 159 cases, (or 62%). An icterus index below 5 is usually regarded as normal. Above 15, one usually sees clinical jaundice; but between an index of 5 and 15 one can spot **latent** jaundice cases which in his series accounted for 53% of the total.

Effective diagnosis of the disease therefore rests with the investigation of suspects along the lines of urinalysis, liver function tests, icterus index, and other serological reactions. Because hepatitis without jaundice is common in the 0 - 4 age group, the risk of spread is therefore great, and its control more difficult. For here the disease can be endemic and unrecognized while in contrast it becomes more obvious in their contacts of older age.

Incubation Period.

The incubation period of the disease varies from 20 to 35 days, but most commonly extends over about 30 days.

It is known that contaminated water held at room temperature for a long period may increase the incubation time[9]. So also will infected water which has been flocculated and filtered[7]. Conversely, when faeces are fed to volunteers, (the only method of experimental passage of the virus for study purposes), the incubation period can be reduced to 20 days. This period is very much shorter than the 120 days observed for its ally, the virus of serum hepatitis.

Period of Infectivity and the Carrier State.

Once the disease is established the period of infectivity is varied. The illness may take a rapid course. Barron's[4] series showed a hospitalization period of less than ten days in 48% of his cases. His suggestion for this was the high number of non-icteric cases among the group. On the other hand, Stokes[8] showed that non-icteric cases in children may have a chronically active hepatitis A.

Two children at the Chicago Orphanage outbreak showed positive stools at 5 and 15 months, respectively, when stool extracts were fed to volunteers. He also described two girls, one of 11 months and another of 28 months whose faeces were positive into the second year of the disease, and neither of whom had showed jaundice[10]. These children were undisputed carriers of Virus A, presumably due to insufficient immunization They did, however, show symptoms intermittently over this period of time, and one can only absolve the case as a source of infection upon clinical recovery.

Prognosis.

The prognosis of the disease is good as far as life is concerned, although case fatality rates cannot be ascertained. The liver appears to make a remarkable recovery from the infection, and only in a few cases is permanent damage noted. Post-

hepatic cirrhosis following established cases of infectious hepatitis have been described[11].

Immunity

One attack of infectious hepatitis is believed to confer immunity for life to that disease. There is no inter-related immunity between infectious hepatitis and serum hepatitis viruses.

Neefe and Stokes described a water-borne epidemic at a summer camp[9]. Four men who had recovered from the disease attributed to a "water agent", were challenged with a "faecal agent" of infectious hepatitis virus. None developed the disease, but nine controls went down with hepatitis. Immunity therefore appears quite rapidly upon recovery.

Possible Routes of Spread.

Various modes of spread have been suggested in the dissemination of the disease, either directly through droplet spray from the nasal pharynx or by an oro-faecal route; or indirectly by means of water, milk, food, flies, etc. In Neefe and Stokes' investigation of the epidemic at a children's camp he describes 572 cases, 344 of whom went down with the disease within seven weeks; in other words, an explosive epidemic of high incidence. Here was a simultaneous infection of individuals who had never yet made direct contact with each other. This being typical of a water attack prompted investigation of the well. The well was surrounded by cesspools from between 75 to 180 feet away, and the water not only showed evidence of faecal contamination, but also produced infectious hepatitis in volunteers. Oral transmission tests on volunteers also showed that the virus was present in the blood, faeces, and serum of cases, including extracts after passage through a filter. The virus was not transmitted, however, in the oral sampling of naso-pharyngeal preparations, urine, or an extract of camp flies caught on the site.

Another case against naso-pharyngeal spread can be made from studies at the Chicago Orphanage[8]. Here the nurses at the Orphanage were going down with infectious hepatitis, while the infants and mothers were apparently free from attack, as already mentioned. Some of these children were, in fact, non-icteric cases of the disease, and the disease was common between them and their nurses. Institution of new nursing techniques put an immediate end to the epidemic, but respiratory contact still existed. Also, the mothers did not get it from the nurses during this intimate contact. This study does however strongly support the feco-oral mode of spread, a method which has been generally accepted in most of many other studies. It is given as a principal mode of spread in the 60 reports of epidemiological investigations received by the United States Public Health Service during the past two years.[3]. In addition to epidemics attributed to water spread, milk-borne, and food-borne epidemics are described.

An outbreak in Forsyth, Georgia, of ten cases, each with jaundice incriminated the milk supply[12]. The cases all occurred within 38 days of each other and all had received raw milk from a local dairy. The total lack of sanitation at the dairy and the occurrence of infectious hepatitis at the dairymen's neighbour together with a gastric upset in one of his own children were supportive evidence of this mode of transmission. Reed et al described an explosive epidemic amongst 24 medical students who ate regularly together at a "fraternity club"[13]. Water, milk and food are therefore without doubt definite vehicles in the spread of this disease. But the experience of most has been the direct transmission within a household. King et al described an outbreak in Richvale, Ontario, of 50 cases of jaundice, 28 of whom all lived within three adjacent streets. Investigation proved them to be cases transmitted by the feco-oral route[14].

Secondary Attack Rates.

Knight[15] et al in a study of 152 cases in Cuppa County, Missouri, found the homes to be the chief centres for dissemination. The schools, however, afford a method of transmission to new family groups. This study is supported by other observers who found the attack rates higher in homes than in schools[6], and who also showed that 10% of the school children who never exhibited jaundice during an epidemic to have physical and laboratory evidence of hepatitis[15].

Lilienfeld et al, working on an outbreak in Baltimore[5], observed 109 cases with jaundice in a housing project. They noticed a secondary attack rate of 8.8 percent of those exposed. Secondary cases are far commoner in families than among outside contacts. This figure is in keeping with other urban studies[16], but secondary attack rates will rise as high as 20 or 30 percent in rural families[17]. They also noted that the administration of gamma globulin reduced this rate to 1.4 per cent significant therefore of the protection afforded by this material.

In view of the fact that secondary cases are so much commoner in families than among outside contacts, and because hepatitis without jaundice is common in the 0-4 age group at least, thus facilitating the risk of spread — we must regard infectious hepatitis as essentially a family disease.

Control.

A temporary control over the spread of this disease can be brought about through the administration of gamma globulin intramuscularly to all contacts of the infection provided it is given at least one week before the expected date of onset[18]. A dose as small as 0.01 m.l., per pound body weight is all that is necessary and provides for passive immunization of the subject[19]. If the contact under such protection still remains exposed

to cases of infectious hepatitis, there is strong evidence that a passive-active immunity may develop, so that the duration of the protection is extended beyond the limits usually afforded by gamma globulin[19]. This fact was made clear in the Chicago Orphanage epidemic already mentioned, when the nurses were given gamma globulin to bring to a close the continuous outbreak that occurred among their numbers. When the gamma globulin immunization program was dropped these nurses remained free of disease in spite of the continuation of infectious hepatitis among the children they were nursing, whereas a new stock of nursing personnel went down with the disease. Because the exact date of exposure is not always known, gamma globulin is often not given a chance to work. Consequently the occasional case will occur among infected contacts. Hsia and others[20] using gamma globulin in the small dose during 1951, injected alternate members of 40 families where there was one case. He gave gamma globulin to 38 adults and 57 children who were established contacts, and only one of these contacts developed the disease. In his control group were 55 adults and 40 children. Secondary cases developed in two adults and 11 children. In 1951 Lilienfeld[5], during a Baltimore outbreak, reported a secondary attack rate of 8.8% of those exposed but only 1.4% when protected with gamma globulin. Ashley[21], using the same small dose, reported only last year secondary attack rates of 15.8% in the unprotected, and 0.6% in the protected groups.

Enough has been said then of the efficiency of gamma globulin in the protection of those exposed sufficient to warrant its use. But it would be poor public health to stop here and rely on immunological methods alone. Stokes[8] said in his management of the Chicago Orphanage study that the institution of better nursing technique brought about an immediate cessation of cases among the new nursing arrivals who were not under any influence of gamma globulin. This not only emphasizes the existence of a previous feco-oral spread and poor technique, but also showed that good methods are enough to curb an epidemic in any hospital or institution where there is an epidemic among the personnel.

As far as communicability is concerned infectious hepatitis should be respected in the same way as typhoid fever and handled accordingly. Adequate sterilization can only be achieved by boiling or autoclaving. Disinfectants are unreliable and the use of paper dishware which can later be burned may save the nursing supervisor many headaches.

In rural areas where a water-borne source is suspected, water should always be boiled. Chlorination of a water supply, leaving a residual chlorine of 1 P.P.M. for thirty minutes is inefficient. Super chlorination with a residual of 15 P. P. M. of chlorine results in definite attenuation of the virus, but does not kill it[7]. Water sterilization with chlorine is therefore of no use.

The freezing of water at -70 degrees Centigrade for many months preserves the virus[22]; thus, ice water from a suspected source is dangerous. The virus withstands a temperature of 56 degrees Centigrade for 30 minutes[22]. This is very close to pasteurization temperatures and infected milk reaching a pasteurization plant may be processed without killing the organisms. Milk control in the case of infectious hepatitis therefore rests at the farm level.

Because it is impossible to determine by history the occurrence of past attacks, we do not know who is susceptible to the disease. Therefore it is not practical to impose quarantine regulations. However, an exception to this rule occurs in the case of food handlers. Because the period of infectivity can only be guided by the clinical occurrence of the disease which may range from a few days to several months, isolation can only be terminated upon clinical recovery. Because future knowledge on the nature of this disease depends upon epidemiological study from careful recording of cases, contacts, and circumstances, these must be carried out in any Institution. This extra trouble will be amply repaid when an effort is made to halt the epidemic and provide material for research. Special vigilance is required by all personnel once a case of jaundice occurs in the district.

Whenever an outbreak of infectious hepatitis is suspected a simple screening test for early and subicteric cases is worth employing.

Bile pigments occur in the urine during the disease often early in its phase of development and their presence in this type of liver disorder offers us good opportunity to spot possible suspects and report them in for further investigation. One such test utilizing Methylene Blue was introduced in 1931[23] and has been used in studies on infectious hepatitis with success[24]. A brief description of this test is as follows:

Methylene Blue Test for Bile Pigments in Urine

Using the standard dropper of 20 drops per c.c., one adds 2 drops of 0.2% aqueous Methylene Blue to 5 c.c's of morning urine. If a green colour is obtained one continues to add the reagent until the blue colour remains constant. If more than 5 drops are required it will be necessary to dilute the urine accurately and start again. Applied to 1,000 normal cases the usual number of drops required of reagent is beiow 5 in number, usually two but rarely more than 4. If 5 or more are required then bilirubin is possibly present. This was observed in 77 cases of infectious hepatitis one week prior to the onset of jaundice by Gellis and Stokes in their use of this test.

Conclusion.

An effort has been made to draw together as many facts as possible concerning the epidemiology of infectious hepatitis, and these facts have been pieced together to provide a clearer picture of our knowledge of the disease at the present time and may serve to guide us in the very difficult problem of its control.

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Medicine

Salt and Water Case Reports Salicylate Intoxication

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Salicylates are widely used by the medical profession and the laity for a variety of ailments. While the incidence of minor reactions is unknown, major intoxications caused by salicylates account for some four percent of all fatal poisonings due to solids and liquids in the U.S.A.[1]. The present report is concerned with those cases in whom serious intoxication is likely, either because of the size of the dose administered or because the drugs were given to an individual already in metabolic imbalance, usually due to fever or dehydration.

The primary disturbance of salicylate overdosage is stimulation of respiration. Deep breathing, which is often rapid, ensues and usually persists. Failure to appreciate that hyperpnea in this situation is a primary event, not a reflection of underlying "Acid-Base" upset, coupled with our lack of knowledge concerning the metabolic effects of salicylates, leads to much confusion in the interpretation of the altered biochemical findings and their proper correction.

Although the picture may be complicated by antecedent disturbances for which the salicylates were prescribed, the usual sequential changes in salt and water metabolism during intoxication are as follows[2-11]:

1 Initially respiratory stimulation leads to a lowering of the carbon dioxide tension in body fluids and a rise in blood pH (respiratory alkalosis). Associated changes in plasma ionic pattern are:

(a) A decrease in the concentrations of HCO_3^-, $HPO_4^=$, K^+ and often Na^+.

(b) A secondary increase in the concentrations of organic anions and sometimes Cl^-.

The urine passed during this period is alkaline in reaction and contains high concentrations of sodium and potassium as the bicarbonate salts.

2. As the duration of the intoxication is prolonged, overbreathing continues and the carbon dioxide tension remains low. Renal loss of cations ($Na+K$) and buffer anions, together with an increase in extracellular organic anion concentration, results in a fall of blood pH values to subnormal levels (mixed respiratory and metabolic disturbances). Associated changes in plasma ionic pattern are:

(a) A continued fall in HCO_3^- concentration.

(b) A continued increase in organic anion concentration which may reach a level of 20 mEq/L above normal.

(c) K^+ may rise to normal or supranormal levels (the extracellular concentration of this ion bears an inverse relationship to pH).

The urine passed during this period is neutral or acid in reaction Its electrolyte composition has not been reported but likely the change in reaction is due to the replacement of the bicarbonate anion by organic anions of lower buffering capacity.

3. Finally, if the situation remains uncorrected, respiratory and cardio - vascular failure ensue. Carbon dioxide tension will rise rapidly with respiratory insufficiency and pH values will fall to low levels. There is likely to be a terminal rise in K^+ concentration.

From the above it may be seen that either alkalosis or acidosis may be found with salicylate poisoning. Where the carbon dioxide combining power method of estimating bicarbonate anion (HCO_3^-) concentration is used, low values will be obtained both during alkalosis and acidosis. In addition, this determination will yield values higher than true bicarbonate ion concentration when overbreathing is present and falsely low

values when underbreathing occurs late in poisoning. Adequate definition of the "Acid-Base" upset in salicylate overdosage can only be accomplished by serial determination of carbon dioxide content and pH or carbon dioxide tension of the blood From any two of these determinations both the upset in hydrogen ion concentration and the extent of the buffer anion disturbance can be found. The following report illustrates some of the difficulties encountered in the management of a case in which these estimations were not done.

9th July 1.00 p.m. A 59-year old Irish laborer was admitted to hospital in semi-coma. In his possession was an empty bottle labelled methyl salicylate. Examination revealed an unresponsive, dehydrated male with a flushed, somewhat cyanotic facies. He was overbreathing; his respirations were deep and rapid (32/min); his blood pressure was 110/65. There was an odor of oil of wintergreen about him. His Carbon dioxide combining power was 8.2 mM/L. Therapy consisted of gastric lavage, continuous oxygen and fluid as follows:

	Na(mEq)	K(mEq)	Cl(mEq)	Cal.
2000 ml M/6 sodium lactate	334			
500 ml whole blood	70	4	55	
1000 ml 10% glucose				400

These fluids were given intravenously over ten hours. No change in clinical status was noted. His urine volume was 240 ml for this period; spec. grav. 1.017, protein 0.03%, sugar 1+, micro 10 to 18 pus cells/hpf.

10th July. The patient was more restless, continued to overbreathe and exhibited some spontaneous muscle twitching.

Serum chemistry:

Na+	144 mEq/L	CO_2 c.p.	15 mM/L
K+	2.7 mEq/L	Cl^-	69 mEq/L

Fluid intake:

1000 ml of a solution containing 80 mEq Na, 36 mEq K, 63 mEq Cl, 60 mEq lactate, Ca and Mg 7 mM.
500 ml whole blood.
1000 ml 10% glucose.

Urinary output: 500 ml.

11th July. The patient was unresponsive and breathing deeply at a rate of 32-38/min.

Serum chemistry:

Na+	136 mEq/L	CO_2 c.p.	20 mM/L
K+	3.3 mEq/L	Cl^-	106 mEq/L
BUN	43 mg/100 ml	Serum salicylates	21mg/100 ml

Urinary output: 50 ml.

12th July. The patient died at 2.00 a.m., having remained semiconscious and continuing to overbreathe until shortly before death. Post mortem examination showed:

1. Edema of brain and lungs.
2. Hepatomegaly and splenomegaly.
3. Tubular necrosis of the kidney.

The combination of hyperpnea and a low carbon dioxide combining power value in the present case was taken as indicating the presence of an acidosis. While no pH determinations were done, the failure to observe clinical improvement following an "alkalinizing" regime suggests that the patient was already in alkalosis and that sodium lactate administration served only to potentiate his metabolic upset. The later appearance of spontaneous muscle twitching and the finding of a low serum potassium concentration in the presence of a low urinary output supports this impression. Some measure of the extent of the metabolic disturbance in this patient is obtained from the chemistry of July 10th. On this occasion total serum cation concentration was near normal when there was a reduction in HCO_3^- plus Cl^- concentration (using CO_2 combining power as an approximation of HCO_3^-) from their usual sum of 130 mEq/L to 84 mEq/L. It is likely that this represents a six or seven fold increase in organic anion concentration. Fluids subsequently given to this patient contained nearly equal concentrations of sodium and chloride with little added buffer anion. In retrospect it would seem that the order of fluid administration should have been reversed, those with sodium and fixed anion (chloride) being given initially when alkalosis is most likely and the sodium lactate reserved for later when acidosis occurs. Obviously, serial determinations of pH and buffer anion concentration would have permitted a more rational approach to the therapy of this case.

The nature of the organic anions found in salicylate toxicity remains unknown[11]. It is likely that their extracellular accumulation is a result of some disturbance of cellular metabolism due either to the direct action of the salicylate ion itself[12, 13, 14], or to the attendant respiratory alkalosis[15]. The changes in extracellular composition in salicylate toxicity are qualitatively similar to those observed in over-breathing alkalosis in normal subjects[16]. In addition much of the cerebral symptomatology, the headache, dizziness, difficulty in hearing, drowsiness and mental confusion are common to both situations. Carbon dioxide inhalations will promptly relieve the cerebral effects of overbreathing alkalosis and prevent the occurrence of cation loss by the kidney and rise in plasma organic anion concentration which are the result of continued alkalosis. While carbon dioxide breathing has been used in salicylate poisoning[8], no studies have yet been reported to indicate its effect where the inhalations have been used continuously in concentrations appropriate to the maintenance of normal carbon dioxide tension and pH values. Such an experiment might well permit a differentiation to be made between the effects of overbreathing alkalosis and the effects of the salicylate ion itself.

Proposed Regimen for Cases of Salicylate Intoxication.

1. Gastric lavage. This is especially important where methyl salicylate has been ingested because of the relatively slow absorption of this preparation. Sodium bicarbonate should never be used as a lavaging fluid because it enhances the rate of gut absorption. The dangers of this procedure in the comatose patient probably outweigh its usefulness.

2. Maintain hydration. This need not await biochemical determinations. Use blood, saline and glucose.

3. Oxygen administration where peripheral circulatory failure is present.

4. Correction of hydrogen ion upset. Where the patient is in respiratory alkalosis (low CO_2 tension and high pH values), continuous CO_2 inhalations may be useful. This should never be attempted in the absence of serial measurements of CO_2 tension and pH. Where pH values are found to be subnormal, sodium bicarbonate or lactate solutions may be used. If pH values are low and CO_2 tensions high, such as may obtain where the respiratory center has failed, artificial respiration is indicated. It should be pointed out that the finding of a low concentration of potassium in a state of respiratory alkalosis is not an indication for potassium administration as the concentration will rapidly rise with correction of the alkalosis. In the absence of biochemical determinations blood, glucose and 0.9% NaCl are the safest fluids to administer when the patient is first seen. Later, if there is no clinical improvement, it is likely the patient is in acidosis and sodium bicarbonate or lactate may be useful.

5. Measures designed for increasing salicylate removal.

A three fold increase in urinary excretion of salicylate will accompany a change in urinary pH from 5.0 to 7.5[17]. A high pH and salicylate excretion will obtain where the patient is in respiratory alkalosis so long as hydration is maintained. Sodium bicarbonate or lactate will do likewise where their administration is accompanied by the formation of an alkaline urine. The use of a carbonic anhydrase inhibitor would likely enhance urinary salicylate excretion. However, its effect on cation (Na+K) and buffer anion excretion would probably outweigh any advantages. Where available, artificial dialysis has been shown to effect reasonably good removal of salicylates[16].

Summary.

Although the specific effects of the salicylate ion on cellular function are largely unknown, the sequence of "acid-base" disturbances resulting from overdosage has been fairly well defined. The plasma CO_2 concentration is reduced at all stages in salicylate intoxication and cannot by itself be used as a guide either to the metabolic disturbance or to the use of corrective therapy. In this situation serial measurements of both CO_2 concentration and pH are necessary for proper clinical evaluation. An outline of therapy in these cases has been proposed.

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Orthopedics

Genu Varum in Children

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Genu Varum or bow legs is frequently met with in practice due mainly to the concern of the parents. Thus, one must treat both the child and the parent, and the latter is often satisfied with nothing less than a diagnosis. This article will briefly summarize some of the causes of bow legs. Diagnostic features and treatment will also be mentioned in each of the entities. The etiology has been divided into the following headings:

1. **Deficiency in Diet**
   (a) Scurvy
   (b) Rickets
2. **Gastro Intestinal Disturbances**
   (a) Coeliac disease
   (b) Fibrocystic disease of the pancreas
   (c) Chronic diarrhea
3. **Urinary Disturbances**
   (a) Renal rickets
   (b) Tubular renal rickets — without acidosis and with acidosis
   (c) Idiopathic hypercalcuria
   (d) Congenital kidney hypoplasia
4. **Infection**
   (a) Osteomyelitis
   (b) Luetic osteochondritis
   (c) Infantile cortical hyperostosis
5. **Congenital Skeletal Defects**
   (a) Congenital tibia vara
   (b) Osteogenesis imperfecta
   (c) Dyschondroplasia
   (d) Kyphoscoliotic tibia
6. **Trauma**
7. **Postural Deformities**

1. Deficiency in the Diet.

(a) Scurvy. This condition is caused by an inadequate intake of water soluble vitamin C. It affects mainly the 6-16 month age group, and is characterized by hemorrhages. Along with the usual signs and symptoms there is anemia, low alkaline phosphatase, and absence of vitamin C in the plasma. X-ray reveals other typical findings such as Fraenkel's line, which is an accumulation of the zone of provisional calcification, the dark scurvy line of Pelkan which is a reactive hyperemia, Pelkan spurs, Wimbergers line or ringing of the epiphyses, a ground glass porosis which incites bowing due to stresses and strains, subperiosteal hemorrhages, and thinning of the cortices. The retardation of growth is due to the lack of adequate bony trabeculae in the final stage of enchondral ossification, due to a disturbance of osteo-apposition, caused by lack of vitamin C. Treatment is administration of Vitamin C and prevention of unprotected weight bearing, till epiphyses apear normal.

(b) Rickets. This is a metabolic disorder causing a defective calcification of the growing bone due to deficiency of vitamin D in the diet. It is classified as foetal, infantile, resistant, and late rickets. Among the usual findings are rachitic rosary, bossing of the skull, craniotabes, widening of the epiphysis, increased alkaline phosphatase, and normal or low serum Ca and P. Roentgenograms reveal broadening of the epiphysis, cupping of the metaphysis, trumpeting of the metaphysis and increased depth of the epiphyseal line due to accumulation of the proliferating cartilage cells plus a deficiency of calcium to form the layer of provisional calcification. Treatment is adequate vitamin D in the diet. Splinting, manipulation, or osteotomy of the long bone deformities may be necessary. Stapling the appropriate side of the epiphysis may be indicated.

2. Gastro Intestinal Disturbances.

At this point one should state that the following gastro intestinal and urinary disturbances produce roentgenograms more or less identical with those seen in rickets:

(a) Coeliac disease. This disease is characterized by difficulty in digestion of fats. There is a resultant loss of the fat soluble vitamins and Calcium becomes saponified by the undigested fats producing a further deficiency in that element. Treatment involves administration of liver and adequate vitamins and minerals in a diet low in fats and starches. Bone deformities would necessitate treatment similar to that of rickets.

(b) Fibrocystic disease of the pancreas—manifests itself in infants and children and can duplicate the skeletal changes of rickets due to the absence of sufficient pancreatic enzymes, and again the loss of vitamin D and diminished absorption of calcium. Treatment is pancreatic enzymes. The prognosis is poor.

(c) Chronic diarrhea—may produce a deficiency of the essentials of bone metabolism, and in the child the rachitic picture is seen.

3. Urinary Disturbances.

Kidney lesions producing bone lesions are classified as Renal Osteodystrophy, and this can be subdivided according to the abnormality of kidney function.

(a) Renal Rickets — is considered a glomerular deficiency whereby phosphates are not excreted by the kidney and there results a decrease in the production of ammonia. Due to loss of ammonia in the kidney, other sources must be called upon to neutralize the acids excreted. Thus calcium is withdrawn from the tissues and from the skeleton in amounts sufficient to produce rachitic findings.

The plasma phosphorus is always raised and some consider that there is a high excretion of phosphorus into the intestine, where it unites with calcium, preventing absorption of the latter. For treatment diets high in calcium and vitamin D have been advocated as palliative measures.

(b) Tubular Renal Rickets — has been a subject of controversy. Dent divides this entity into types according to the deficiency of the tubular absorption. As far as therapy is concerned, there are two main types.

In the group which does not produce acidosis is Fanconi's syndrome, characterized by hypophosphatemia, aminoaciduria, and intractable rickets. Here the phosphates are lost in the urine, being robbed from the skeleton, and causing an associated loss of calcium. This accounts for the high calcium and phosphorus in the urine. The amino-acid hydroxyproline, which is abnormally excreted, is considered to make up about one third of collagen, another essential entity in osteoid production. Thus, there is a deficiency in osteoid deposition, again inciting rachitic deformities. Treatment is high doses of vitamin D and a good diet.

The group producing acidosis is called renal acidosis, in which there is defective tubular function with respect to urinary acidification and ammonia formation. Fixed bases such as calcium and potassium are utilized to neutralize the plasma hyperacidity. With a body loss of these two ions, rachitic conditions may result from the former and familial periodic paralysis from the latter. The child appears acidotic with neutral or alkaline urine. Treatment of mild cases should include administration of sodium bicarbonate. Severe cases require high calcium, potassium and vitamin diets.

(c) Idiopathic Hypercalcuria — is characterized by an excess excretion of calcium in the urine. The body need can be compensated by adequate dietary intake.

(d) Congenital Kidney Hypoplasia — may also produce metabolic bone deformities through the calcium and phosphorus upset secondary to kidney disfunction.

4. Infection.

(a) Osteomyelitis — or bone infection secondary to trauma, may affect the growth of the epiphyseal plate, if the infection is in that region. Normal enchondral ossification may cease in consequence of the inflammatory reaction to the infective organism. Thus, an epiphyseodesis may be produced, and if the lesion is on the medial aspect of the epiphyseal plate, genu varum will result. This would tend to be unilateral in most cases. For treatment successive open wedge osteotomies may be necessary for adequate correction, and the other leg may be a candidate for stapling.

(b) Luetic Osteochondritis — due to congenital lues, is a condition occurring in the first year of life. The spirochete attacks the epiphyseal plate, disrupting the proper development of the zone of provisional calcification, producing an irregular edged area of increased density in the region of the metaphysis. There is an associated bilateral periostitis. The epiphyseal distortion may lead to bow legs. Following the institution of anti-luetic treatment, this growth disturbance reverts to normal-appearing bone. The same osteochondritis in the shoulder is known as Parrot's pseudoparalysis, because the arm frequently lies flaccid by the side.

(c) Infantile Cortical Hyperostosis, rare as it may be, is another condition which frequently shows bending of the tibia. It is mentioned here, as it is considered to be caused by a virus. In this entity there is excess periosteal bone laid down, followed by porosis of the underlying cortex. The resultant bone is weakened, with accompanied bowing due to stress factors. The infection runs its course in 6-12 months, and the bones as a rule correct themselves.

5. Congenital Skeletal Defects.

(a) Congenital Tibia Vara or Osteochondrosis Deformans Tibia — is a condition of unknown etiology, the varus deformity becoming obvious at various ages of the growing child. Radiologically it is characterized by a triangular epiphysis with the apex medial, and a beak-like projection of the proximal medial tibial metaphysis. There is faulty and delayed ossification of the medial portion of the involved epiphysis. Treatment of the mild conditions is correction of foot strain by wearing proper shoes with appropriate wedges. Advanced cases warrant osteotomy, or stapling of outer tibial epiphysis with proximal fibular epiphyseodesis.

(b) Osteogenesis Imperfecta — This entity, characterized by multiple fractures, frequently has tibial bowing. Here the zone of ossification is deficient, with defective evolution of osteoblasts, resulting in few, small, delicate, formed trabeculae, with a fibrous type of tissue intervening. The resultant bone is very weak.

(c) Dyschondroplasias — such as Morquio's disease, are mentioned here, as they also involve the epiphysis, producing long bone deformities.

(d) Kyphoscoliotic tibia — is a congenital deformity of the tibia. The bowing occurs in the distal part of the middle 1/3 of the tibia, and may occur in various directions. As a rule the deformity presents antero-lateral bowing and X-ray reveals a characteristic ossified mass at the concave side of the curve. The lesion is primarily in the diaphyseal centre of ossification. Surgery should be delayed as long as conservative therapy appears beneficial.

6. Trauma.

Fractures involving the medial lower femoral or upper tibial epiphyseal line may produce genu varum due to arresting growth in the area of the

lesion. Other forms of trauma such as epiphyseal slipping, epiphyseal cartilage trauma, and surgery in that region, may cause growth disturbances to produce a similar deformity.

7. Postural Deformities.

At birth there is normally some internal tibial torsion, bowing of the tibia, and increased anteversion of the neck of the femur — all of which gradually correct themselves with growth of the child. However, certain sleeping and sitting habits can be prime factors in augmenting these various deformities of the lower extremities in children.

Internal tibial torsion causes a pigeon-toed stance or posture such as in No. 2. This appears as a bow leg deformity, but on examination one can estimate the degree of internal rotation present in the tibia by palpation of the medial and lateral malleoli, with the knee at 90 degrees. Roentgenograms frequently show the tibia to be straight. Positions augmenting this deformity are the tailor position as shown in No. 4, and sleeping in the knee-chest position with the lower extremities as in No. 4. The above positions also tend to increase any actual bowing of the tibia which may be present since birth. Increased anteversion of the neck of the femur also predisposes to a pigeon-toed and seemingly bow leg posture. It can be diagnosed when there is 5-6 plus internal rotation of the hips as compared to 2-3 external rotation. This element is enhanced by the reversed tailor position with the feet externally rotated as in figure No. 3. This position, although creating internal rotation hip contracture, will tend to correct internal tibial torsion, as does the frog sleeping position in No. 1. From the above, it is apparent that the examination of bow legs must include the pelvis to the toes.

Treatment of these postural defects will include:

(a) In infants, institution of proper sleeping habits, i.e. sleeping on the side.

(b) In children, development of proper sleeping and sitting habits.

(c) Torsional stretching exercises.

(d) Denis-Browne boot night splints. The toe should be cut out of the shoes.

(e) Proper shoes. These should support the foot in the anatomical position. Outside sole wedges may be of cosmetic value but may be detrimental to the foot.

(f) Osteotomies including derotation and/or wedging of the tibia or femur, or both.

(g) A more than adequate vitamin D intake.

Conclusion

Bowing of the lower extremities in children should be investigated as follows:

(a) History to estimate dietary conditions, health of the child, familial incidence, and sleeping and sitting habits of the child.

(b) Complete physical examination.

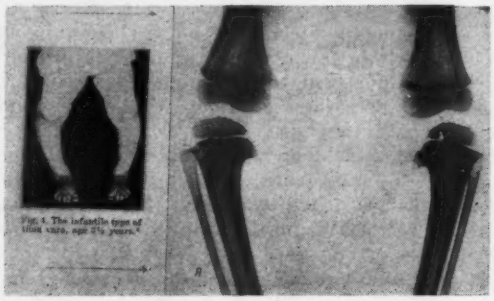
(c) An A-P roentgenogram on a 14-17 inch film to include pelvis and lower extremities, with the patella facing anteriorly.

(d) A proper laboratory investigation if the above does not diagnose the condition.

Summary

A classification of conditions giving rise to bow legs is presented.

A short note on characteristic features and treatment of the various entities is included.



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Psychiatry

Some Community Aspects of Mental Deficiency

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The amount of mental deficiency in the community has been variously estimated at from 0.5 to 3.0 per cent. Surveys have been carried out from time to time in different countries to ascertain what proportion of the population was likely to be mentally defective. One of the first comprehensive surveys, carried out in England and Wales, was the subject of the Wood Report in 1929[1], which established a defective rate of 8.6 per thousand for the population as a whole, with a higher rural incidence of 10.4. The Scottish Mental Surveys of 1932[2] and 1947[3], dealing with the intelligence of eleven-year old school children, established the fact that 1.5 per cent could be regarded as mentally retarded. Samplings in the United States have varied as high as between two and three per cent, an average estimate being 2.2 per cent given by Wechsler[4].

Further light is shed on these divergent estimates by Penrose's analysis of the 1929 Wood Report[5]. Taking the incidence of defectives by age groups he showed that although the overall incidence was 8.6 per thousand, it was actually 30 per thousand at 12 years of age, due to the rigid standard of the scholastic environment, but only 5.7 per thousand in the 30-39 age group, wide choice of employment favoring adjustment. Where higher estimates of mental defect have been obtained they have been based essentially on intellectual criteria. Mental deficiency. however, is more than merely intellectual retardation. It is, properly speaking, a state of both intellectual retardation and social incompetence, resulting from arrest or incomplete development of mind, occurring at birth or from an early age. On this basis, therefore, only a proportion of children with an intelligence within the defective range could be expected to develop, in addition, the social incompetence necessary to justify certification as mental defectives. Dealing with American children Wallace Wallin[6] gave it as his opinion that not more than 0.5 per cent would ultimately become mental defectives in the true sense of the word. It seems probable that over the population as a whole one per cent at most would come within the scope of mental deficiency. The number of cases requiring institutional care is still less, being commonly estimated at 2 per 1000 of the general population.

Clinically, mental defect is associated with a wide range of conditions and the number is steadily growing. Not so long ago the list would have been made up of a major group of simple primary defect and a numerically smaller group of about seven special types, like hydrocephalic and cretin. In the course of time simple primary has acquired additional names; the terms endogenous, familial, aclinical and subcultural are used more or less synonymously. Lewis[7], who coined the word subcultural, divided defectives into subcultural and pathological groups.

In drawing attention to the prevalence of the former in conditions of pauperism and slumism he pointed out a correlation between cultural conditions and subcultural deficiency. Such defectives, essentially of moron and high imbecile grade, blend almost imperceptibly with the dullard section of the community. Subcultural defectives were regarded by Lewis as representing intellectually the lower range of normal variation of the population.

Both genetic and environmental factors, however, have been postulated in its etiology. Genetically it may depend on the additive effects of multiple genes. Environmentally it is equally clear that an unsuitable and unstimulating background must also play a part, not only in contributing to an antisocial or asocial pattern of behaviour, but also in preventing the individual reaching his potential limit of intelligence. Indeed it is just this factor which holds out the main prospect of improvement, for by replacing such a background at an early age our institutions may best hope to rehabilitate. Due allowance must therefore be made for environmental factors. Actually the tendency is now growing to stress the environmental rather than the genetic component, and this is understandable, for environment after all can be altered whereas heredity is relatively immutable. Nevertheless, in the current swing of the pendulum there is perhaps danger that we may do less than justice to the influence of heredity.

The pathological group of defectives has steadily expanded during the past twenty years with the demonstration of a significant correlation between mental defect and an ever-widening range of physical conditions. We now know that about eighty conditions are associated with mental defect. These include relatively uncommon entities like dyschondroplasia, arachnodactyly and cephalofacial angiomatosis, as well as the more frequently occurring diffuse brain injury. Significant correlations have been made, moreover, in the field of metabolic aberrations where diseases like diabetic exophthalmic dysostosis and gargoylism have joined cerebro-macular degeneration as causes of amentia. Another instance is provided by phenylpyruvic oligophrenia. Previously children with

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this rare metabolic error were just part and parcel of an undifferentiated group. With the discovery of their failure to metabolize phenylalanine a distinctive disorder was recognized and a new disease added to the pathological group. Through increasing knowledge and more refined methods of investigation it is highly probable that other conditions will be similarly isolated.

Nevertheless the hard core of defectives, and the largest single group, remains the subcultural. For this reason any demonstrable increase in subcultural deficiency would be of some significance to the community, irrespective of the relative proportions of heredity and environment in its causation. The suspicion that an increase is occurring has indeed been voiced on more than one occasion. Apart from increase due to better diagnosis and ascertainment the major causes put forward have included differential fertility and the mounting complexity of civilization.

Higher fertility in the lowest social groups such as problem families, is one reason put forward. That such groups are more fertile is quite clear. Amongst other investigations a comprehensive study of problem families in an English city showed that this group had exactly double the average birth-rate for the city[8]. A survey of a similar group in Sweden revealed four times the average birth-rate[9]. A possible factor in this increase may conceivably be, as some have claimed, the humanitarian measures of the modern state.

So far as environment is concerned, efforts to change it by provision of new housing areas have proved no solution and the tendency is for these families to drift back towards the worst slums. Low economic level and irregular employment have been cited, yet on the same or lower income it can be shown that their neighbours are able to maintain decent homes. In such cases it becomes difficult to escape the conclusion that irregular employment is due to inefficiency and poor adaptability of the wage-earner.

One factor which constantly emerges from studies of problem families is the high incidence of mental subnormality in the parents, especially the mothers. In a series of 89 mothers tested by Savage, no fewer than 70 per cent were of subnormal intelligence[10]. Indeed it has been estimated that in about one-fifth of such mothers intelligence is within the defective range[11].

It has been said that "intelligence may be regarded as the cause of economic status only if opportunities are equal and competition is entirely free"[12]. It would appear that these criteria are nearest fulfillment in Britain of today where the social system has led to an extensive levelling of population, and where full employment has provided more jobs than there are workers. And yet, surveys held there have not been free from disquieting interpretation. One of the most extensive reviews was the 1947 Mental Survey of Scottish school children, in which a group intelligence test was given to 70,805 children whose eleventh birthday fell in that year. In itself this showed no falling off in intelligence, compared with a similar survey carried out fifteen years earlier. It did, however, show that in general low scores were obtained by the children of large families and high scores by children in small families. On consideration of the available evidence the view was advanced that familiarity with the material might have produced spuriously high test results and that in reality these results might conceal an actual fall.

In 1952 the official organ of the Scottish Department of Health had this to say: "..... it is only too clear from the Registrar-General's statistics that there is a differential birth rate, highest in the lowest social classes, and lowest in the managerial and professional group; and a differential birth rate in an age of equality of opportunity means in effect that we are breeding from the least intelligent and least socially efficient stock and failing to breed from the best."[13]

A different interpretation was made by Sir Cyril Burt[14]. In summarizing the existing evidence at the request of the Royal Commission on Population he suggested that some compensatory mechanism of a genetic nature might be at work to counteract the adverse effects of the differential birth-rate. This theme was further developed by Penrose[15], notable for taking a less gloomy view of differential fertility. Penrose put forward the suggestion that members of intellectually inferior groups might be as fit biologically as their apparently more favoured neighbours. As the genes whose additive effects determined the level of intelligence were, presumably, spread over the population, the increased fertility of the subnormal groups might be part of a biological compensatory mechanism. By providing a reservoir of intelligence genes they might, according to Penrose, make good the loss of genes resulting from the relatively low fertility of the highly intelligent.

The obscurity is perhaps lessened a little by what we know of assortative mating. This is the tendency for like to marry like. People tend to choose partners whose physical and mental traits resemble their own, and it would appear that similarity in intelligence between husbands and wives is one of the highest associations to be recorded. Concerning the quality of the children of such unions a further glimmer of light has been shed by investigators like Cattell[16], who showed that the average intelligence scores of both parents resembled the man of the siblings in the family. Here again it would be difficult to exclude entirely the respective effects of a stimulating or dull intellectual climate. But in actual practice any increase in associative rather than random

mating would, presumably, lead to an increased scatter of intelligence and a tendency, as pointed out by Slater and Woodside[17], to accentuate extremes in the population.

It may seem paradoxical to consider the possibility of increasing mental defect in these days of rapid scientific progress, and, if we should countenance the suggestion, we prefer to do so in terms of atomic hazards. And yet, there may be more prosaic causes than cosmic rays.

If there is really a risk of such an increase from differential fertility, it would be reasonable to enquire whether there is anything to suggest the operation of this factor in the past. For an investigation of this nature it would not be amiss to consider the example of older civilizations. An instructive example is Ancient Rome, whose final imperial phase offers a suggestive picture. In the high birth-rate of the lowest social group, the very name of which indicated that all the state could expect from it was offspring, there would appear to be clear evidence of differential fertility. The latter days of the Empire were characterized by a physical and mental degeneracy hitherto unknown. The German historian Seeck was maintaining as far back as 1910 that this degeneracy was above all the fundamental cause of the decline of Rome[18]. While most Historians would favour a more composite explanation ample indications have nevertheless come down to us of falling physical standards. Greatly struck by this feature Han Gunther wrote: "In Homer, Thersites is the only cripple; in the late Roman writers long lists could be made of bodily deformities and signs of degeneration"[19]. Argument based thus on the past is apt to be more or less speculative, but it would appear a reasonable assumption that an increase in physical defect could not be divorced entirely from, at least, some rise in mental defect.

Less controversial and conjectural is the effect of increasing complexity of civilization in swelling the ranks of high grade defectives. There can be little doubt that the greater intricacy of modern life is showing up as defective many who were capable of independent existence under less complex conditions. Penrose[20] goes so far as to say that high grade and borderline mental defect are conditions which have assumed prominence only with the development of urbanized and industrialized society. In earlier days, before mechanization began to pervade all aspects of existence, a wide range of simple crafts flourished in the community. Individual craftsmen had not yet given way to factories and assembly lines, and the homelier, more personal supervision of the old order still held sway. Simple work in a less complex society provided lowly, but steady and secure employment for the subnormal.

The coming of a more industrialized order has brought new problems for the subnormal. The effect of the changing situation can be seen in the not uncommon case of the man who is able to carry on in quiet rural employment, but who fails to make good on transferring to an urban industrial environment. Drifting from job to job, and finally breaking down in a delinquent or neurotic fashion, he is referred for examination and found to have an intelligence within the defective range. As the legal criterion of mental defect depends on "ability to manage himself and his affairs with ordinary prudence", it is clear that this individual of impaired intelligence was not obviously defective in a simple rural setting, but became so on exposure to more complex urban industrial influences. With the mounting intricacy of our civilization it therefore seems inevitable that the ranks of high-grade defectives will be swelled by the operation of this factor in itself. Such an admission does not necessarily signify an entirely bleak outlook, although it does imply a change in the attitude of the community towards the subnormal. Admittedly, there is greater difficulty in fitting defectives into a modern setting, but with patience and understanding, adequate training and supervision, and additional safeguards like hostels, they can still contribute happily and usefully to the community.

Finally, to recapitulate, it is apparent that high estimates of 2 or 3 per cent mental deficiency in the community are based on intellectual criteria alone, whereas the number of individuals who display in addition the requisite social incompetence is much smaller.

The actual figure is probably somewhere between 0.5 and 1.0 per cent of the population whilst the proportion which is sufficiently defective to require institutional care is still lower, in the region of 2 per 1000 of the general population.

Nevertheless it does seem probable that an increase in mental defect is occurring, whether from differential fertility, increasing complexity of our civilization, or both.

Such an increase is bound to be felt in the medical, educational and sociological fields, and its implications make it highly desirable that we accord it due weight in the sphere of mental hygiene, and, above all, in the realm of research.

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Children's Hospital, Winnipeg

Ward Rounds

Edited by Wallace Grant, M.D.

Special Guest: Professor M. M. Wintrobe
University of Utah, Salt Lake City, Utah
Chairman: Dr. Harry Medovy

Case I.

A case of microscopic hypochromia anemia in an infant age 6 months.

Dr. B. Devlin: This infant was admitted because of vomiting, pallor and anorexia for the last two to three weeks. She is a female with a normal birth history and a normal prenatal history as far as her mother is concerned. Born full term and breast fed for the first three months. Following this she was given pablum, fruit and strained chicken. No added vitamins were given. She was quite well until the age of 5 months, at which time she developed occasional vomiting after feeds with loss of appetite. On two occasions there was the question of slight blood staining in the vomitus. In the middle of this month the symptoms became worse and she vomited almost every feeding. The vomitus consisted only of milk. Increasing pallor has been noted during the preceding month.

On examination she seemed reasonably well nourished, but was obviously pale. There was no fever. Weight was 14 lbs. Developmental status was normal. There are no petechiae on the skin and in fact the rest of the examination was quite normal.

Laboratory investigation showed the following:

Hemoglobin — 4 grams
R.B.C. — 3,800,000
Reticulocytes — 5%
Packed cell volume — 21
M.C.H. — 14
M.C.V. — 55
M.C.H.C. — 20%
Sedimentation Rate — 6 mm.
Platelets — 318,000
Polymorphs — 16%
Eosinophils — 2%
Basophils — 1%
Neutrophils metamyelocytes — 4%
Lymphocytes — 72%
Monocytes — 5%
Marked hypochromia, polychromasia and an isocytosis.
Plasma proteins — 4.9 grams %
Serum albumin — 3.9 grams %

Serum globulin — 1.0 grams %
Serum bilirubin — Direct — 0.21 mgm.%
Total — 0.42 mgm.%
Stool for bilirubin — negative.
Thymol turbidity — 1 unit.
Thymol flocculation — negative.
Cephalin cholesterol flocculation — ++.
Stool examination — two examinations. No occult blood.
Chest film — negative.
X-ray Longbones: No evidence of scurvy or rickets.
Blood Group — Group A Rh +. Mother's Group AB Rh +.
Coombs Test — negative.

Report by Dr. Hoogstraten on Blood Smear— There is a noticeable left shift in granulocytes. There is a very noticeable degree of anisocytosis. The large erythrocytes appear well filled with hemoglobin, while the smaller cells exhibit a moderate degree of hypochromia. Poikilocytosis is moderate.

Barium enema — negative.
X-ray esophagus, stomach and duodenum — fails to reveal any congenital malformation or evidence of ulceration.

Dr. Wintrobe: One aspect of the presentation, Dr. Devlin, is I think, very pertinent from the standpoint of medical practice. You commented on everything except the appearance of the blood smear, then I was surprised to hear the pathologist being asked to comment on that. Do you not examine the blood smear yourself?

Dr. Devlin: Quite frequently we do, yes.

Dr. Wintrobe: It seems to me that in this case, the most pertinent part of the physical examination of the patient, is the examination of the blood smear. I may say that this morning I had a chance to look at this child's blood smear and it

is the most important part of the whole story. If it is not the practice of the doctor who has charge of the patient to look at the blood smear, and if it is common practice to leave that to the pathologist, then I think the doctor is making a very serious mistake. I cannot understand why examination of the blood smear is so frequently overlooked by the doctor himself. It seems to be true in most parts of the world for the person who holds the stethoscope seems to be afraid of a microscope. There may be some psychological reason for this. As medical students we used to think that all the microscopic work was a gripe and a lot of work and we couldn't quite understand why we had to do it all, but so many times one sees that the report on the blood is made by a pathologist of doubtful competence in the field, sometimes by a technician who knows very little about it and cares less, with the physician not taking the trouble to look at the blood smear himself. The same physician may have a report on x-rays made by a person specially trained in that field and yet he will often want to look at those x-rays himself, even though in theory he doesn't know as much about it as a radiologist. Or he would want to look at the electrocardiogram himself, even though he doesn't know as much about it as the physician who is reporting the case. Unfortunately every hospital does not have a Dr. Hoogstraten, and I am making such a point of it in this case because the blood smear is so perfectly typical.

The smear I saw was evidently taken on admission and it showed very few of these large, well filled cells that Dr. Hoogstraten described. At that time the smear showed many small, pale, poorly filled red cells, and that is the characteristic feature. The calculation of Mean Corpuscular Hemoglobin Concentrates are simply confirmations of that. The important thing is to look at the smear. The indices can be wrong and all sorts of people have difficulty making red cell counts, and hemoglobinometers can be off. But if you have a good blood smear there isn't much you can do that will lead you into error so that the appearance of the blood smear is very important and essential as part of the examination of the patient. In this particular patient it is the tell-tale part of the story because obviously on looking at this child's blood smear it has a microcytic hypochromic type of anemia.

Now, under what circumstances may one get a microcytic hypochromic type of anemia? Well, by far the most common cause is iron deficiency. It is true one gets a similar type of anemia in association with Thalassemia—but there one would expect to see more target cells. There were very few target cells in the smear I saw. Experimentally one can produce a microcytic hypochromic anemia with pyridoxine deficiency, and this is now being done and has been seen in infants, but even in instances where the deficiency has been deliberately produced, the anemia has not been a marked feature of the disturbance. So that the finding of a smear like this 95 times out of 100 means iron deficiency. Then the question comes up, why should there be iron deficiency? Looking at this child, seeing its generally good condition except for pallor and lack of any other physical findings would make one suspect that it had not been getting sufficient iron. As you well know the infant is born with a certain supply of iron derived from the mother but that supply is not sufficient to take care of the requirements for growth. Growth is of course the important factor here. As one person puts it "The growing child bleeds into its rapidly expanding blood volume". The red cells are broken down and the iron is used over again but the blood volume expands so much that the amount of iron is insufficient, so that, unless there is an external source for the young infant and growing child, iron deficiency anemia can be expected. This does not usually happen by 6 months of age but is more apparent at 9 to 12 months or even 18 months of age. One of the unusual features in this case that I would like to hear Dr. Medovy comment on is why this child should have developed iron deficiency anemia at this early age. The other interesting feature is that it has been getting adequate amounts of milk, and judging by its nutrition it has presumably had adequate amounts, yet its plasma proteins are so very much reduced. I presume that examination has been repeated and this is a correct figure.

Dr. Devlin: It has not been repeated.

Dr. Wintrobe: Well, I presume you have the kind of laboratory that you never question. I hope you do. Unfortunately I am a sceptic. I know if someone reports something in a physical examination, I like to check it myself. I see no more reason to give the laboratory any more credence than physical examination or anything else. Mistakes sometimes happen and it never hurts to recheck. But perhaps your laboratory is so well organized and running so beautifully that you never expect mistakes. If this is a correct figure I would want to know why this child has developed hypo-proteinemia. One of the reasons for the x-ray examinations, I presume, was to determine whether there is some explanation for this. One wonders on finding such an anemia at 6 months whether there might actually be some abnormality here, some lesion whereby this child is actually losing blood. Apparently. there is no ulcerative lesion in the gastrointestinal tract. This makes the matter all the more puzzling. Is there no history of dark stools to suggest melena? Another matter worth commenting on is the fact that the bone marrow was examined in this infant. I don't

know whether this was done simply as a matter of interest or whether it was thought that it might be helpful in diagnosis. If it was the latter, I would beg to differ. I think, by and large, in the study of patients with blood disorders bone marrow examination is done much too often and, as I have already implied, examination of the blood smear is done much too little. In this case, from a standpoint of diagnosis, I don't think one could expect very much from the examination of the bone marrow, and I personally don't like to stick a needle into a child's bones unless there is a very good reason for it. In cases of iron deficiency anemia I don't know any good reason. If one is dealing with leukemia, in certain special circumstances, it might be necessary. So I think we will have to leave the diagnosis there Dr. Medovy, that this child has iron deficiency anemia, and one can expect that iron therapy will relieve the anemia. Dr. Hoogstraten's report on the blood smear yesterday sugests that there is already regeneration. There are now not only the small pale, poorly filled red cells which were present originally, but there are now some rather large, well filled cells which are the young cells coming out and that is further supported by the report of the reticulocyte count which has risen to 16%. In short, the blood is responding to the iron therapy. This is an iron preparation given by mouth, and I think this is worth commenting on because it is as often abused in practice.

It is common for an infant with this condition to be transfused. I believe that this is a mistake. Those who give transfusions only occasionally live in blissful peace. I guess. But those of us who see patients who are transfused often do not have such peace. We run into patients who have had trouble from blood transfusions. Blood transfusion is not a perfectly innocuous procedure, as you well know. There are many objections to giving blood transfusions and it should be done only when there is a good reason. In my opinion iron deficiency anemia is not a good reason. One can give the iron by mouth. Some people are a little too impatient. One can't expect a response within 2 to 3 days. It takes a little time.

This child has been on treatment for 6 days, and there is already a response which is good enough and I don't see why one needs to be impatient with such a good reticulocyte response already evident. However, the hemoglobin cannot be expected to rise for awhile yet. That will take 2 to 3 weeks from the time treatment is started. This is a physiological response. This is bone marrow now making cells because it is now getting a substance which hitherto it has lacked. This is the kind of response we want. There isn't really any hurry. The child may shock us when we see a hemoglobin report of 3 - 4 grams, but it is we who are shocked and not the child. The child has been developing this anemia very gradually and his cardiovascular system has become adjusted to this deficiency of hemoglobin. The child could be shocked by the transfusion, but it certainly will not be shocked by the report from the laboratory.

It is important to appreciate that these children should be treated by iron rather than by blood transfusion. One might argue under certain social and economic circumstances there might be some advantage in giving iron intravenously. We could then give the baby the amount of iron that we know that it requires to bring its blood back to normal and letting it go at that. However, in most cases the iron can be given orally and the response, although gradual, is quite satisfactory and intravenous iron is rarely necessary. I merely stress the fact that it would be preferable to use the intravenous iron rather than a blood transfusion in this case, if we were in a hurry for some special reason. No matter how neglectful a family may be the mere fact that they have finally brought the child for medical care indicates that they have become worried and they are likely to follow the orders when it comes to giving a child some fluid substance to take three times a day.

Dr. M. Berger: I wonder if there is any relationship between the low serum proteins and nutritional anemia.

Dr. Medovy: This patient was presented because he was a puzzle. This type of anemia in an infant so young is uncommon. The coincident finding of low serum protein is in my opinion also uncommon. This has led us to wonder whether the anemia in this instance is due to blood loss rather than to ordinary nutritional factors. We have not done protein estimations on too many of these babies but I cannot recall others who have had low serum proteins. Dr. Israels, can you recall any in the last few years?

Dr. Israels: I can recall two cases that we have seen in recent years, and it is possible that if we look for them we will find them.

Dr. Medovy: The main reason we asked for the serum protein determination in this case was the unusual circumstances that the baby was 6 months of age with a reasonably good feeding history who showed a low iron type of anemia.. I am afraid the bone marrow examination was done for the same reason.

Dr. Wintrobe: May I ask Dr. Israels how he would explain the hypoproteinemia? He says that we will find it if we look for it which I think is probably true, but how do you explain the development in a child taking an adequate milk diet?

Dr. Israels: I think this child has enough likelihood of blood loss to account for the anemia. The fact that we did not pick it up on two stool

examinations does not mean that he hasn't had some previous blood loss and maybe some current blood loss. The vomiting of substances that seem to have blood in them is significant and yesterday there was some red streaking on the stool which may have been blood. If the baby is in a precarious situation at the start, in other words, depleted of the iron stores the mother gives it and fed on milk so that the intake of iron is not very great, it wouldn't take much of a loss to put him in the condition we find him in even at 5 or 6 months of age.

Dr. Wintrobe: Do you think it might be an ulcerative lesion of the gastro-intestinal tract? In spite of the negative x-ray report?

Dr. Israels: Well, that is possible. We did think that we saw blood in the stools and the mother reports that there has been some vomiting of reddish material. As for the type of lesion I haven't the foggiest idea, and that's why we asked for barium swallow. I thought it might be the lower end of the esophagus.

Dr. Wintrobe: Do you get peptic ulcers at this age? You don't really think that the child has some psychological problems?

Dr. Israels: Dr. Childe tells us that there is no active ulcer now.

Dr. Medovy: We have been interested particularly in the problem of hiatus hernia. Several of these cases have been picked up in Toronto and Sawyer has published the facts about these cases. All of them had vomiting with occasional hematemesis. We have had no luck so far in picking up hiatus hernia in infants here. We are certainly aware of the condition and we are trying to pick them up. So far the x-ray evidence is nil. We have also been interested in trying to find a case of peptic ulcer since in Pittsburgh they seem to be able to find about 20 cases a year. Perhaps however, the emotional problems in Pittsburgh are greater than the ones in Winnipeg.

Dr. Wintrobe: If you have ever been in Pittsburgh you would understand why.

Dr. Medovy: We have certainly not had much success in finding peptic ulcers in infants and children. There is another condition which I hope Dr. Chown might say something about and that is the feeling that some of us have that some of these babies lose blood at the very moment that they are born. They develop a non hemolytic anemia due to blood loss and this blood loss occurs sometime at the moment of birth. They actually bleed into the mother or bleed during the process of being born. This may be due to some abnormality of the placenta or tearing of the placenta or tearing of vasae prebae, and these babies may start off with a lower margin than we realize. They may leave the nursery on the eighth day with a hemoglobin down to 10 or 11 grams. It is not an easy condition to prove unless the bleeding has become massive. As you know Dr. Chown did manage to prove it in a case of mine about a year ago. This was I believe, the first instance of the proof that a baby could bleed into the maternal circulation. Investigation would have to be carried out on the day of birth in order to establish beyond doubt that this phenomenon had occurred. I see no reason why this type of blood loss occurring in a milder degree might not set the stage for this type of anemia. In other words the blood loss would not have to be gastro-intestinal either by vomiting or by melena but could be by direct blood loss due to fetal bleeding through the placenta or into the maternal circulation.

Dr. Wintrobe: I think this is a terribly interesting subject. I would like to hear about Dr. Chown's further studies. I read his paper with considerable interest as I read all of his papers because I know that there is going to be something in them.

Dr. Chown: We have not been able to confirm any further cases. I understand Dr. Day of New York has three further cases of bleeding into the maternal circulation. It is certainly a possibility here but would it account for the low serum protein.

The Editor's Summary:

This has been a case of microcytic hypochromic anemia in a 6 month infant. The factors involved have been discussed by Dr. Wintrobe and other members of the staff. No definite decision has been made as to whether this bleeding could be acounted for entirely on the basis of dietary deficiency and in view of the diet this baby received. It would seem that other causes were operating. Two possibilities here were considered. One was that this baby, in fact, had lost some blood from the gastro-intestinal tract at a point of ulceration which could not be demonstrated. and secondly that this baby might possibly have lost blood during its first few minutes after birth as a result of some placental defect or as the result of tearing of fetal blood vessels. The baby's response to oral iron was satisfactory enough that she was discharged from hospital. The reason for the low serum protein value is still not clear.

Tuberculosis

Tuberculosis Yesterday, Today and Tomorrow

F. W. Jackson, M.D.
Director of Health Services, Ottawa, Ont.

Everyone in this audience who was born and lived in Canada before the beginning of the present century must have vivid recollections of the toll in sickness and death taken by tuberculosis around 1900.

The town in which I was born and brought up was no different from any other small town in Canada in that it was a rare thing indeed to find a family going through a period of twenty years without illness or death caused by the tubercle bacillus; and, in many instances, families were almost decimated by this disease.

I remember the young mother who was found to have advanced pulmonary tuberculosis following the birth of her first child. Although she took the then prescribed "cure" — rest in bed while running a temperature—she proved a source of infection to her subsequent family of eight, and four members were dead before I reached adult life.

In those days "consumption" and "galloping consumption" were talked of only in whispers and young ladies were said, in awed tones, to be going into "the decline", a euphemism employed by our parents when we youngsters were within hearing. However, we grew to know what the term meant, and though the name "phthisis", mentioned by the sick English chap with the strong Cockney accent, gave us a little more trouble, one wise young lad on hearing that the gentleman must get all the fresh air he could, said, "Oh, he's got galloping consumption" and our word power was increased forthwith.

When I attended Medical College here (1908-1912) I saw more of the ravages of tuberculosis. Many of the cases were single men, laborers, who when taken in were accommodated in a temporary ward at the General Hospital. Tuberculosis among the well-to-do was treated in their homes.

At the beginning of the century there appeared on the list of students enrolling in theology at Manitoba College, and later at the Medical School, the name of a man destined to be widely known as an expert in tuberculosis. I speak, of course, of the late Doctor D. A. Stewart.

In 1908 we had the beginning of an organized program for the control of tuberculosis in this Province, with the formation of the Sanatorium Board. It was the result of three or four years of intensive persuasion and education carried on by Dr. Stewart with Governments — provincial, city and municipal—and with voluntary groups. Trying to do 24 hours work a day took its toll, and Doctor Stewart fell prey to the very disease he was dedicated to fight, necessitating nearly two years of treatment at Saranac Lake. Finally, enough money was collected to make a start on the Sanatorium at Ninette, and it was a proud day for Dr. Stewart and his confreres when that Sanatorium opened its doors for the reception of patients in November 1910. Fortunately, Dr. Stewart was able to come back from Saranac Lake to become its first Superintendent.

Two years later, the City of Winnipeg opened the King Edward Memorial Hospital to take care of tuberculosis.

After my graduation in 1912, I had the good fortune to set up practice in Wawanesa, a town between Ninette and Brandon. My first introduction to Dr. Stewart was on a train that travelled to Brandon. When the conductor of the train was taking my ticket he said, "Dr. Stewart from Ninette is on the train. Have you ever met him? He is a fine man." I said that I had not, and he took me back and introduced us. Our friendship lasted from that day until Dr. Stewart died, some 24 years later—a long and cherished period of time for me.

I have many happy memories of Dr. Stewart and his kindness to many of my patients.

Certainly he did much in our part of the Province to stimulate good medical practice. He had many impromptu medical meetings, especially if he had an interesting case or two. I doubt if there was ever a Manitoba Medical Association meeting that he did not make a request to have some part of the meeting held at Ninette Sanatorium in order to demonstrate to the medical profession the facilities available to treat tuberculosis.

For many years he used the Sanatorium as a training school for medical students from Manitoba Medical College, and I firmly believe that one of the main reasons so many cases were discovered by the general practitioners in Manitoba a few years ago was the excellent training they received at Ninette under Dr. Stewart.

Dr. Stewart was particularly interested in trying to ensure that there would be ample accommodation for tubercular patients. With this end in view, in 1927 he requested the Provincial Government, which then had as its Minister of Health and Public Welfare, the late Dr. E. W. Montgomery, to make a study of health conditions in Manitoba, with special emphasis on tuberculosis.

Read before the Annual Meeting of the Canadian Tuberculosis Association, June 8, 1955.

It was one of the highlights in my career to be asked by Dr. Montgomery, in the Spring of 1928, to undertake this survey of the Province under the auspices of the Welfare Supervision Board. This study resulted in the recommendation that there should be a Central Tuberculosis Clinic established in the City of Winnipeg, and that another sanatorium be situated somewhere in the vicinity of that city.

The Central Tuberculosis Clinic, alongside the Winnipeg General Hospital, was opened in 1930, and shortly afterwards, in upper St. Vital, on a beautiful site on the Red River, the St. Boniface Sanatorium opened its doors to receive patients.

With the addition of these new beds, and the increased number of people under treatment, the load of keeping track of all the known cases, treated and untreated, their contacts in the community, and the dates at which they should be brought in for check-up, became more than could be carried around in the head of the public health nurse whom the Department of Health and Public Welfare had seconded to the Central Tuberculosis Clinic. So in 1937 the Central Tuberculosis Registry was established.

The operation of the Registry under Miss Elsie Wilson does more, I think, to stimulate the orderly examination of known cases and contacts in the community than any other single thing that has been done in this Province.

Dr. Stewart was never content to sit still. He was always after somebody for something to help in the tuberculosis program. He obtained assistance from the Provincial Department of Education who employed school teachers to work in the Sanatorium and assist young people to continue their education. This started the Sanatorium staff —as well as some of the patients—thinking about rehabilitation, and in 1942 the Sanatorium Board started such a program. This, with the recreation program available at Ninette Sanatorium, now goes a long way to meet the needs of the patients, both by occupying their time advantageously while they are in the institution, and also by helping them to be suitably placed after their discharge.

One cannot leave the subject of tuberculosis of yesterday in this Province without paying a tribute to the Sanatorium Board of Manitoba and the excellent work it has done. The time and energy put forth by every member of the various boards since the first one was formed 46 years ago constitute a monument of self-sacrifice. This, as well as the work of other voluntary agencies, has contributed much to the well-being of the people of this Province. I was fortunate to be a government-appointed member of that Board for a period of nearly twenty years, and it is a privilege today to express my personal appreciation of the Board's tremendous contribution to Manitoba's tuberculosis control program. Voluntary effort is needed in all worthwhile endeavours, because, without it, there is lacking that public knowledge and support so essential to effective action.

So much for yesterday's tuberculosis program! What about today?

We have available in this country, certain fundamental resources that are of the greatest importance to a good health program, particularly in the control of tuberculosis. We have in Canada the second highest standard of living in the world; we have better housing than nearly any other nation, and most important, we have the means of providing adequate nutrition for all our people. Insofar as being able to do a good job is concerned, we are in my opinion, the best situated country in the world.

The special resources and facilities we have can be listed under four headings:

1. Treatment
2. Case finding
3. Research
4. B.C.G. Vaccination.

The building up of facilities and resources over the last ten years — and particularly under the stimulus of the National Health Grants Program —has provided all the provinces with the extra requisites needed to round out and fill in gaps in their respective programs. In fact, two of our main resources, namely treatment by drugs and case-finding through hospital admission X-ray examination, are almost one hundred per cent paid for out of the Grants. Dr. Wherrett, in a paper published in the March issue of the Canadian Journal of Public Health, was kind enough to say:

"Federal participation has come when it is most effective. With such assistance, the case-finding program has been extended greatly and treatment services have been brought up to the peak of efficiency. The newer drugs have been made available on a wide scale, and facilities for chest surgery extended. There is no doubt the Federal grants have played a great part in the results obtained."

Canada now finds herself in the happy position of having, as at the end of 1954, the third lowest death rate of any country in the world. The number of new cases found each year seems to be on the decline, so that today we stand on a firm place to look forward and plan for tomorrow.

I am convinced that each of us in the public service (and I include, of course, those working the field of tuberculosis control) must continue to hold fast to the determination to achieve the greatest good for the greatest number of people. To do this, one must try to plan ahead and have in sight some specific goal. In the field of tuberculosis, our aim must be to use to the best of our ability the ample facilities which we now have available, in order more quickly to achieve our goal — the elimination of this disease as a major menace to mankind.

Sometimes one is able, when one gets outside a specific field, to see the problems in a different light, and to suggest general lines of attack; so I am going to take the liberty of telling you in broad terms, what I think we should do.

(1) Treatment

(a) Beds

The indications are that, if the trends evident in the last few years continue in reported deaths and new cases, we have enough beds. This is demonstrated in this Province by the fact that the City of Winnipeg has been able to close the King Edward Memorial Hospital and use the building for more pressing needs.

(b) Drugs.

We hear some rumours that, with the new and effective drugs now being used, more patients are being treated outside of sanatoria. In my opinion, this is entirely wrong. When beds are available, every person who is sick with tuberculosis should be under routine sanatorium care. Home treatment for an infectious or potentially infectious disease should not be tolerated in any enlightened society.

(c) Surgery.

More and better surgery, I think, will provide quicker and more lasting cures, and no doubt the next four or five years will see the present techniques greatly improved to take care of most infected lung tissue.

(2) Case Finding

(a) Hospital Admission X-ray Program.

Although this has been a most effective method of finding new cases, I do not think that we have yet achieved our maximum possibilities in this respect. Some hospitals appear to consider that routine X-ray of all admissions presents too many difficulties. In spite of this we should continue to strive by every means we have to ensure that 100% of the patients admitted to all hospitals have a chest film at least sometime during their stay in hospital.

(b) General Surveys.

For the last two or three years now I have questioned the value of general mass surveys, especially since, as Dr. Wherrett has pointed out, these are largely supported from Grant funds. My own opinion is that we should take a good look at community surveys to make sure that some redirection is not indicated.

(3) Research

I think that our research program should be considered by some appropriate body, and a long-term plan outlined so that we can inaugurate a program to try to obtain more information about this disease. This is especially important in respect to the use of B.C.G.

(4) B.C.G. Vaccination

Although we have had B.C.G. vaccination for many years, we do not seem to be able to lay our hands on any reliable statistical data by which to judge its effectiveness. There is evidence that certainly B.C.G. vaccination of persons most directly exposed to the disease, namely nurses and doctors, gives them a better chance of going through their period of exposure without infection.

Personally, I see, in going through the literature, more and more mention made of B.C.G. vaccination, particularly in some European countries. On many occasions one picks up a medical journal from Great Britain and notes where a County Council Medical Officer has a program going in the school medical service, whereby every child at school leaving age, who has a negative tuberculin test, is vaccinated with B.C.G. Perhaps this particular method might be something worth considering in Canada.

I would earnestly suggest to you that we take a new look at our approach to the finding of sources of infection. Why can we not use the same methods as we have always used in our general public health work in controlling other communicable diseases?

This means the integration of tuberculosis control epidemiological facilities with those of health departments at the federal, provincial and municipal levels of government. Once a case of tuberculosis is diagnosed it should be followed back until the **original** source of infection is found. It does not matter how long it takes, or what energy it requires; this is the only way, in my opinion, that we will finally get control of this disease. This requires, I think, a combination of the intensified Venereal Disease control efforts used during the War with the usual methods for case finding of typhoid, diphtheria, or any other transmissible disease.

I am sure the problem of re-admissions has bothered every sanatorium superintendent. Some very valuable experimental work might be done in this connection by the greater use of well trained medical social workers, who, before a case is ready for discharge, would have the responsibility of interviewing the patient, investigating the environment from which he came, in order to try to make sure that the patient is going to fit into his old environment without the possibility of breakdown. This may mean many things from a social viewpoint; such as housing, nutritional standards, family relationships, income, and dozens of others, and it may be necessary that certain alterations in the environment would have to be worked out before the patient could be discharged.

In conclusion, Mr. Chairman, Ladies and Gentlemen:

To our host for this meeting, the Canadian Tuberculosis Association, which has served us and the Canadian people for some fifty-five years, all of us owe a debt of gratitude which none of us

can pay. Its activities reach every part of Canada, and its educational work with the public at large has had much to do in placing us in such a satisfactory situation in our work in the tuberculosis field.

To Dr. John Wherrett who has served as its Executive Secretary for nearly a quarter of a century, we say from our hearts: "Thank you, John".

We at Ottawa depend on Dr. Wherrett's advice for decisions we have to make in our Grants administration. Hardly a day passes that I, or someone else from our staff does not phone his office. As often as not, we find he is in Newfoundland or British Columbia, on the Prairies, or in the Maritimes—anywhere in this great country of ours, or in Rome, France or England, wherever his expert advice may be needed. I would like to say, Dr. Wherrett, on behalf of the Minister, the Honorable Paul Martin, Dr. Cameron the Deputy Minister, and myself, "Thanks for all you and your Association do for the Department of National Health and Welfare."

Twenty-three hundred years ago Archimedes, in explaining the operation of the lever, said: "Give me a firm place on which to stand, and I can move the earth." I would suggest that, as a result of your endeavours over the past few years, you now have a firm place upon which to stand. I believe we can look forward to the day when tuberculosis, which has been the scourge of mankind since the beginning of time, can be moved, by the combined efforts of all of us, to a position of no greater importance as a menace to health and happiness of the Canadian people than typhoid or diphtheria is today. If I may be excused for paraphrasing a famous saying of Sir Winston Churchill, I suggest to you that we have the tools —let us go forward with confidence and finish the job.

Abstracts from the Literature

Association of Irradiation with Cancer of the Thyroid in Children and Adolescents: D. E. Clark, J.A.M.A., 159: 1007, 1955 (Nov. 5).

15 cases of cancer of the thyroid were seen in children 15 years of age and younger. All 15 had previous x-ray therapy for benign conditions about the head, neck, and thorax. The time from irradiation to the diagnosis of cancer of the thyroid averaged 6.9 years. The total dose in air ranged from 200 to 725 r. This observation lends support to the idea that an association exists between irradiation and the subsequent development of cancer of the thyroid in late childhood and adolescence. The increasing incidence of cancer of the thyroid between 1900 to 1950 in patients under 15 correlates with the increased use of x-ray for enlarged thymus, tonsils, and adenoids. Previous irradiation about the neck in early life may be an etiological factor in development of cancer of the thyroid in late childhood and adolescence.

A. G. Rogers.

ABO Blood Groups and Gastric Acidity: K. H. Koster, Erik Sindrup, Vagn Seele. Lancet, July 9, 1955.

A comparison was made in the distribution of ABO blood-groups in patients with carcinoma of the stomach, gastric ulcer, or duodenal ulcer, with the distribution in healthy persons living in the same locality.

Previous reports show the amount of free acid in the stomach ranges from the highest to the lowest values as follows: duodenal ulcer, gastric ulcer, normal, carcinoma of stomach with free acid, carcinoma of the stomach with achlorhydria.

Distribution of A B O blood groups in 413 patients with carcinoma of the stomach were studied. 51.3% were group A, compared with 44% of controls. The presence of free hydrochloric acid was studied in 301 of these patients. 195 patients were achlorhydric, and of this group, 56.4% were blood group A, and 33.2% were blood group O.

Distribution of blood groups in 1047 patients with proved peptic ulcer were studied. Blood group A occurred in 39.3% compared with 44% of controls, and against 56.4% in patients with gastric carcinoma and achlorhydria. Group O occurs in 48.7% compared to 40.6% in the controls. There is a greater prevalence of group O in patients with duodenal ulcer than in patients with gastric ulcer (50.2% compared with 46.3%).

The shift from conditions associated with the least production of hydrochloric acid is accompanied by an increasing frequency of blood group O, and a decreasing frequency of blood-group A.

S. Malkin.

The Blood Groups in relation to Peptic Ulceration and Carcinoma of Colon, Rectum, Breast and Bronchus. An Association between the A B O Groups and Peptic Ulceration. Ian Aird, Ch.M., F.R.C.S., J. A. Mehigan, M.B., B.Ch., B.Sc. and J. A. Fraser Robers, M.D., D.Sc., F.R.C.P., British Medical Journal, August 7, 1954.

A survey was carried out at 12 hospitals in England and covers cases treated during the years 1948 - 53. Rigid criteria of diagnosis were used. For the carcinomas, only cases proved by histological report on material obtained by biopsy, operation, or post-mortem examination, were accepted. The majority were surgically treated. In peptic ulceration, the criterion was a macroscopic report (operation or gastroscopy), and the majority of cases were perforated or bleeding ulcers, or those submitted to elective surgery. This series does not include many medically treated cases, and therefore is not representative of all patients suffering from peptic ulceration.

13,000 case records were scrutinized and of these, 7,702 were satisfactory as regards diagnosis and record of blood group.

Knowledge of associations between diseases and blood-group frequencies is at an early empirical stage. There is as yet no prior expectation that a particular disease will show an association.

There were 3,011 patients with peptic ulcer, 2,599 with cancer of the colon and rectum; 998 with cancer of the bronchus; 1,017 with cancer of the breast.

Persons of group O are about 35% more likely to develop peptic ulceration than are persons of the other groups.

The ABO frequencies shown by the three cancers do not differ significantly from the population controls.

There is no significant difference in the proportion who are Rhesus negative.

S. Malkin.

Football Hematuria. A. W. Boone, E. Haltiwanger, R. L. Chambers, J. A. M. A., 158: 1516, 1955 (Aug. 27).

Gross hematuria was noted in 6 of 37 football athletes in the 1954 season. The incidence was at its peak after each game. Microscopic hematuria may accompany strenuous conditioning exercises. Both gross and microscopic hematuria cleared with rest. If restrictions of activity because of hematuria had been carried out, the entire team would have been benched, as all players had microscopic hematuria.

A. G. Rogers.

Aldosterone Observations on the Regulation of Sodium and Potassium Balance: J. A. Luetscher Jr., R. H. Curtis. Annals of Int. Med., 43: 658, 1955 (Oct.)

The adrenal gland secretes aldosterone. This decreases sodium excretion, and increases potassium excretion. The aldosterone output in normal men is related to sodium and potassium intake, and suggests that the output is an effort to return altered balance to normal. Aldosterone output is increased in (a) normal men on low sodium or potassium diets, (b) in the nephrotic syndrome, congestive heart failure, hepatic cirrhosis or toxemia of pregnancy when edema accumulates, and in (c) tumors of the adrenal cortex which secrete aldosterone. Patients with adrenal cortical tumors with hyperaldosteronism showed hypokalemia, alkalosis, hypernatremia, with severe muscular weakness, periodic paralysis, tetany resistant to calcium therapy, hypertension, little or no edema, and signs of mild chronic renal insufficiency. "Potassium-losing nephritis" is now considered "primary hyperaldosteronism." A. G. Rogers.

The University of Manitoba Institute of Psychiatry Meetings March 19th and 20th

Meetings will be held in the Medical School Auditorium, Winnipeg.

The program will be of particular interest to psychiatrists, but other interested physicians are invited to attend.

Registration fee is $2.00.

Registration forms and a copy of the program may be obtained by writing, The Director, Department of University Extension and Adult Education, University of Manitoba.

Medical Library Evening Hours

Sponsored by

The Winnipeg Medical Society

The Library will be Open from
8 p.m. to 10 p.m.
Monday through Friday
from
February 6th, 1956
Regulations

(1) The Library Committee wishes it understood that the Closing Hour of 10 p.m. will be STRICTLY ADHERED TO:

(2) All Reading Room facilities available to Physicians and Students;

(3) The Student on duty will assist in looking up subjects in the Quarterly Cumulative Index Medicus for the last ten years;

(4) If previous references are required they should be obtained during the regular library hours (9 a.m. to 5.30 p.m.).

(5) **The stockrooms will NOT BE OPEN.**

The Medical Library Committee.

Our President 1955 - 1956

Ruvin Lyons was a graduate from the University of Manitoba with the degree Bachelor of Arts in 1928. His medical studies were carried out at St. Bartholomew's Hospital, London, England, and he was qualified member of the Royal College of Surgeons, England, and Licentiate Royal College of Physicians, London, in 1934. He continued postgraduate studies and became a member of the Royal College of Obstetricians and Gynaecologists in 1939. During World War II he served with the Royal Canadian Army Medical Corps. He became certificated in the specialty of Obstetrics and Gynaecology, Royal College of Physicians and Surgeons of Canada, and is an associate on the staff of the General Hospital, and a member of the University of Manitoba, Faculty of Medicine. In 1955 he was elected a Fellow of the American College of Surgeons.

A man of many interests, Ruvin was elected Treasurer of the Manitoba Medical Association at the Annual Meeting in 1949. He held that office until 1952, and the following year was elected Second Vice-President. The office of First Vice-President to which he was elected in 1954 is usually classified as "non-arduous" but with the resignation of Reg Whetter in 1954, a heavy load was thrust upon the broad Lyons shoulders when he assumed the title of Acting President. So capably did he carry out the duties that at the Annual Meeting in 1955 he was elected to the highest post in the gift of the Association. Ruvin is also a member of the Board of Trustees, Manitoba Medical Service, and a member of the Advisory Committee under the Health Services Act. He is ably supported in his duties by his charming wife Edith and cherishes the affection of a son and daughter.

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Editorial

S. Velsrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

What's In A Name?

Medical nomenclature abounds in eponyms. Diseases, syndromes, therapeutic procedures, laboratory methods, radiologic and electrocardiographic findings, and even procedures of physical diagnosis, have a generous share of them. Indeed, we should be thankful that some descriptive terms still remain. Would it not be frightening if, for instance, we were to refer to percussion as the Augenbrugger manoeuvre, or speak of doing a Laennec when we auscultate the chest as glibly as we speak of doing a Babinski when we stroke the sole of the foot?

Eponyms in Medicine, as in other branches of human endeavor, are primarily intended as a tribute to the discoverer, the inventor, the pioneer. Commendable and understandable as this desire to give credit where credit is due may be, it often leads to complications. For one, there is frequently a lack of unanimity on the matter of the party deserving of the "credit". While, for example, in English speaking countries the name of Graves graces the diagnosis of hyperthyroidism, on the Continent of Europe it is that of Basedow.

Sometimes one gets around this difficulty by giving credit to all claimants. Marie and Strumpel, Tay and Sachs, Albers and Schoenberg, may be resting better in their eternal repose in the happy knowledge that none of them has been slighted, and that their names properly linked grace the pages of respectable medical texts. The disadvantages of this solution, however, are only too apparent. How, for instance, is one to tell that, while Marie and Strumpel are two unrelated persons, Argyll-Robertson is the name of but one man? Or how is one to credit a large number of contenders? It is bad enough to have the names of Parks, Christian and Weber attached to a syndrome without being reminded by someone in the know that it was really Kalischer who first described it. One wonders as to the number of correct diagnoses missed because of the diagnostician's subconscious reluctance to get stranded in a morass of unpronounceable names.

It is, thus, easy to see that too many cooks spoil the nomenclatorial broth. What may be less apparent is the opposite evil of one cook having his finger in too many terminological pies. One name may be found on the label of several diseases. Pick, for instance, has his name tagged on to two neurological disorders as well as a disease of serous cavities. Pick, of course, is but a name picked at random. Von Recklinghausen, Charcot, Weber and others guilty of the same offence come readily to one's mind. They have all contributed generously to the quota of sleepless nights of earnest medical students.

An ingenious innovation in the field of eponyms, which could easily, if widely accepted, obviate all difficulties is the one introduced by the resourceful group of investigators, who named a newly discovered coagulation accelerating factor — the "Christmas" factor. This name has no seasonal implications, nor is it the name of the doctor who discovered the factor, but that of the patient in whom its absence was first observed. For the first time in history the patient was getting credit for something he did (or rather, did not) possess. A step farther in the same direction was taken by the virologists who named a strain of the virus of Poliomyelitis after Brunhilde, the monkey in whom the strain was first isolated. Whether, in keeping with this trend, we are about to witness a wholesale bestowal of names of pet rabbits, guinea pigs or mice upon diseases and syndromes, is difficult to foretell.

It was only to be expected that the difficulties attendant upon the use of eponyms, as well as the paucity of information conveyed by them, would lead to an outcry against their use, and an increasing demand for their substitution by descriptive terms. Indeed, the pendulum is now swinging in the opposite direction. It is, accordingly, no longer fashionable to speak of Graves' disease, or that of Von Recklinghausen's. The more informative Thyrotoxicosis and Osteitis Fibrosis Cystica are preferred.

Yet it is worth noting that the use of eponyms is not without its advantages. If not too cumbersome or name-laden, the eponym is an abbreviator and time saver. How else, indeed, could the numerous structural abnormalities of the tetralogy of Fallot or Eisenmenger's Complex be condensed in one word? Would it not be a blessing if we could substitute a convenient (preferably monosyllabic) eponym for such ponderous descriptive names as Agnogenic Myeloid Metaplasia, or Lupus Erythematosus Disseminatus Acutus.

It is also possible that the main weakness of the eponym — its lack of informative content with regard to pathology or etiology may, at times, be a source of strength. It may, occasionally, be advantageous to have a name that is non-committal when we are dealing with a condition, the nature of which is none too certain. A case in point is that of Polyarteritis Nodosa. This disease, characterized by inflammatory involvement of all vessel walls, carried for years the name of Periarteritis, with its misleading implications of periarterial inflammation only. A convenient eponym might have obviated the confusion.

Let us, then, not discard our crutches until we are able to walk more confidently.

Eponyms are here to stay . . . at least for a while.

Convocation 1956

The University and the Medical School

On the evening of Friday, January 20, a special convocation of the University of Manitoba was held in the new wing of the Medical College. The corner stone of the $750,000 structure had been laid by the Lieutenant-Governor on the previous afternoon in weather 20° below zero, but on the Friday evening the building was ablaze with lights which shone on academic garb and evening dress. The convocation was an intimate occasion which emphasized the relation of the Faculty of Medicine to the University as one of its integral parts.

With appropriate ceremony the President of the University opened the Convocation. Three candidates for the degree of Doctor of Laws, honoris causa, were presented to the Chancellor: Dr. G. D. W. Cameron, Ottawa, Deputy Minister of National Health, by Dean Lennox Bell; Dr. A. F. Menzies, Morden, by Professor Colin Ferguson, and Dr. R. B. Mitchell, Winnipeg, by Assistant Professor C. H. A. Walton. It was pointed out that Dr. Menzies and Dr. Mitchell had been president of the Manitoba Medical Association. The Chancellor, Victor Sifton, C.B.E., D.S.O., LL.D., conferred the degrees "with all the rights and privileges pertaining thereto".

Dr. Percy Johnson, Flin Flon, president of the College of Physicians and Surgeons of Manitoba, presented to the University on behalf of the College a cheque for $11,000 for the furnishing of the new Library, and Dr. A. R. Birt, Winnipeg, President of the Winnipeg Medical Society, presented a gift for the furnishing of the Doctors' reading room. The Medical Male Voice Choir, "the Lennox Bell Singers", sang three fine songs under the direction of Keith Christie, 4th year Medicine.

Dr. Cameron in his scarlet doctoral robe delivered an address in which he mentioned the medical care given by the Department of National Health to Canadian citizens, especially Indians and Eskimos, and the aid given to far Eastern nations. He paid tributes to Manitoba medical graduates both outside and within his Department; in particular Doctors M. R. MacCharles, J. C. Wilt, F. W. Jackson, Percy Moore, J. W. Wood, Cameron Corrigan and Lynn Falconer.

The relation of the physicians in Manitoba and the University has not always been so close. The University began in 1877 as a federation of three denominational colleges— St. John's, Manitoba and St. Boniface. Wesley College came in later. Up to 1899 the University was merely an examining and degree-conferring body, and it was not till 1904 that it operated in a building of its own, the old Science building on the Broadway site.

In 1883, under pressure from would-be medical students, Manitoba Medical College was founded. The first classes were held in the Central School and in a cottage on Harriet Street. The College was founded on faith, for in a little city of 25,000 inhabitants set down on the prairie, five hundred miles from any large centre, and still smarting from the collapse of a real estate boom, many forecast only a short life for the institution. Yet the members of the faculty were young and able, the students took hardships in their stride, and both had the western outlook of hoping that the next year would be better.

One of the principles insisted on by the thirteen incorporators headed by Dean James Kerr was that the granting of degrees should rest solely with the University of Manitoba. Thus began the affiliation of the College with the University. From the beginning the College was never a diploma mill. The members of the teaching faculty were not paid— on the contrary, they put their hands in their own pockets and with other subscriptions from lay friends, had a solid brick building erected in 1884. This still stands at the corner of McDermot Avenue and Kate Street. In 1906 the first unit of the present Medical College was opened.

In 1918, through the wise and unselfish policy of Dr. H. H. Chown, then Dean, the Medical College become an integral part of the University as its Faculty of Medicine. The property and equipment of the College, free of debt, was transferred to the University.

If the shades of those early teachers, James Kerr, R. J. Blanchard, A. H. Ferguson, J. W. Good and J. R. Jones, had revisited the medical school on the evening of the convocation they would have witnessed "the substance of things hoped for, the evidence of things not seen". Their faith had prevailed.

Association Page

Reported by M. T. Macfarland, M.D.

Letters To The M.M.A.

January 31, 1956.

Dear Doctor:

The following letters have been received from the General Secretary of the Canadian Medical Association:

January 24th, 1956.

"I attach for your information a copy of a letter which accompanies our distribution of certificates of attendance at the B. M. A. - C. M. A. - O. M. A. meeting in 1955. Since the letter was drafted, the judgment of the Exchequer Court in the appeal of Griffith vs. M. N. R. has been handed down and it dismisses the appeal with costs. This decision is, of course, adverse to our interests and we now depend on the outcome of our representations to effect an amendment to the Income Tax Act.

Despite the unfavourable decisions and the uncertainty of appropriate legislative action, it is the opinion of the Income Tax Committee that entitled physicians should be advised to claim convention expenses incurred in 1955 in their income tax returns for that year. They should be prepared, however, to have such claims disallowed if all efforts to have them recognized should fail.

I report this matter to you because you may feel disposed to issue certificates of attendance at Divisional Annual Meetings under the same conditions and because you will be required to answer enquiries from members who are uncertain how they should proceed."

February, 1956.

"Attached please find a certificate of your attendance at the 88th Annual Meeting of this Association in June 1955. For the years 1948 to 1954 inclusive practising physicians were permitted to claim in their Income Tax Return, as an expense against income from professional fees, the reasonable expenses of attending:

a) One Convention of The Canadian Medical Association.

b) One Convention of a Provincial Medical Association.

c) One Convention of a specialist society in Canada or the United States

but a ruling of the Income Tax Appeal Board has disallowed these claims. This ruling has been the subject of an appeal to the Exchequer Court of Canada but the judgment of the Court has not yet been handed down.

Your Association has made the strongest recommendations to the Honourable Minister of Finance, that an amendment of the Income Tax Act be made to establish and to extend the right of doctors to deduct the expenses of attending medical meetings, and we are hopeful that such amendment may be passed at the current session of Parliament.

There is, however, no ruling available at this time which actually authorizes such deductions. Under the circumstances, practising physicians who incurred expenses in attending meetings of the three types enumerated above during the year 1955, are advised to claim for such expenses in their Income Tax Returns. It is possible that such expenses may be disallowed but if they are not claimed in 1955, it is doubtful if they would be admitted in subsequent Returns.

Members may be assured that every effort is being made to regularize this matter of convention expenses and to have recognized meetings further afield than the three types originally authorized. Until an authoritative ruling is available it is recommended that 1955 Returns be made in the manner outlined."

"P.S. Since the above was drafted the judgment of the Exchequer Court has been announced, dismissing the appeal. This decision is adverse to the deduction of convention expenses."

Yours very truly,

M. T. MACFARLAND, M.D.,

MTM/ea Executive Secretary.

♦

December 23rd, 1955.

Mr. H. J. Seed, President,
British Pacific Insurance Company,
535 Homer Street,
Vancouver 3, B.C.

Dear Mr. Seed:

The Executive of the Manitoba Medical Association has requested me to write you concerning the information recently forwarded by your company to the doctors of Manitoba.

We are very pleased to learn that the company is interested in providing a form of prepaid medical service. As you may know this is consistent with the principles of the Canadian Medical Association in its attitude towards the protection of individuals and advocates where possible voluntary care of medical expenses in a prepaid manner.

The Executive, however, wishes to make clear to you that our profession should not give any greater co-operation to your scheme than it does already give to other commercial carriers in this field. Thus it is felt that if your subscribers are sold their contracts in the belief that the full cost of their medical care is covered by the fees noted in your schedule, it is a presumption on your part and may produce considerable dissatisfaction on the part of the patient.

It is noted that your fee schedule appears to be a proportion of a minimum fee schedule, based upon an agreement between members of the medical profession in Manitoba, whereby low income citizens in our province may obtain protection at a figure they can afford. In this instance, in order to provide such services, the Manitoba Medical Service has been established. This differs from a commercial carrier in that it is a non-profit organization and its medical members are voluntarily absorbing the loss accruing from the very comprehensive service they provide. The doctors, however, should not be expected to co-operate with commercial interests, at such a loss to themselves, to provide such coverage as is available through the Manitoba Medical Service.

Therefore, Mr. Seed, we wish to advise you that we will welcome your subscribers but will advise them when they present the Identification Card you mention that they will be liable for the difference which may exist between the amount of their coverage and the charges that generally exist in private medical practice. Furthermore, in order that the members of our profession may understand their position in this situation, a copy of the communication will be forwarded to them as well as being published in the Manitoba Medical Review.

Yours very truly,
M. T. MACFARLAND, M.D.,

Nominations to the M.M.S. Board of Trustees

As a result of nomination at the Annual Meeting and mail ballot, the following were nominated to serve on the Board of Trustees, Manitoba Medical Service, for a term of three years, 1956 - 1959:

Doctor Cecil William Clark
Doctor Donald James Hastings
Doctor Abraham Hollenberg

Grateful appreciation of the Association is extended to the retiring members:

Doctor C. E. Corrigan
Doctor A. M. Goodwin
Doctor M. R. MacCharles

♦

Economics Committee Report

The following is an excerpt from the Economics Committee Report presented to the Executive Committee on December 14th, 1955, following a meeting of the Canadian Medical Association Committee on Economics in Toronto December 1st and 2nd:

"D.V.A. Fees

"It was reported that the D.V.A. through the Treasury Board of the Federal Government had raised fees for office, home and night calls to doctors to $3.00, $4.00 and $5.50 respectively. The usual annual total of these calls is 47,000.

"Relating to the request of the C. M. A. that D. V. A. raise all procedure rates 20%, it was repeated that this had been turned down. However, a motion was passed that as certain charges were markedly low on the D.V.A. fee schedules, an effort to improve specific inequities should be made."

Future Events

The Manitoba Medico-Legal Society

The Annual Meeting of The Manitoba Medico-Legal Society will be held during April or May, 1956, at the Manitoba Club. This meeting will be preceded by a reception and dinner.

At a meeting of the Society to be held on Tuesday, March 27th, 1956, at 8.15 p.m. at The Medical College, Mr. W. J. Johnston, Q.C., Senior Crown Attorney of the Manitoba Attorney's General Department, will deliver a paper on the subject "Some Medico-Legal Aspects of Crime".

The annual dues permit the annual reception and dinner to be held at a very reasonable cost to the individual members. Enquiries may be addressed or dues forwarded to the Secretary, 208 Power Building, Winnipeg. All Manitoba doctors and lawyers are requested to assist by their attendance and participation in making this year's meeting a success. Further notices will be sent to all enrolled members of the Society.

Children's Hospital

Winnipeg, Man.

Re: Ward Rounds and Clinical Conferences

1. Weekly Grand Round 11 - 12 a.m. Thursday mornings throughout the year.

2. Medical Staff Clinical Luncheon, the first Friday of each month (except July and August), 12.30 to 2 p.m.

3. Special Tuesday noon conferences 12 to 1,
   First Tuesday of the month, Therapeutics, (Dr. Nickerson).
   Second Tuesday, X-ray Diagnosis, (Dr. Childe).
   Third Tuesday, Cardiac Conferences, (Drs. Ferguson, Medovy, Armstrong, etc.).

All these meetings take place in the Playroom at the East end of the first floor.

The members of the Medical profession are invited to attend these Conferences and Ward Rounds.

189

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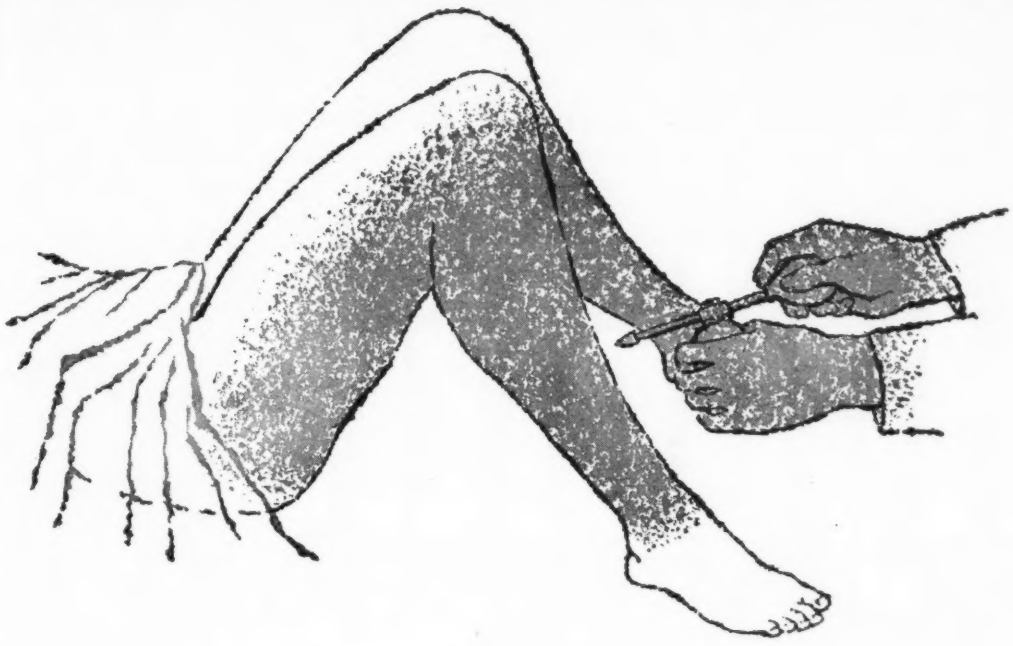

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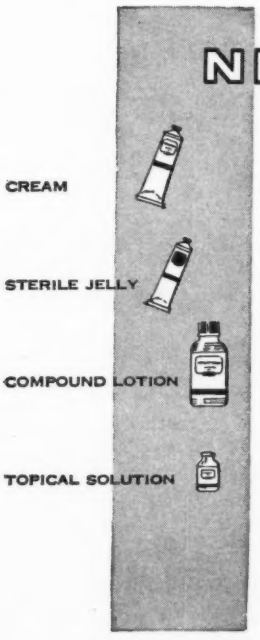

Peal and Karp's impressive report[1] on 858 trials of this new topical anesthetic, includes 100 cases of hemorrhoidectomy, repair of fissure in ano, and anal ulcer.

Neither toxicity nor sensitization was observed. Yet "in comparing the post-operative pains of those patients who had previous rectal operations, the comment was made that TRONOTHANE 'gave more satisfactory relief than any other drug used previously.' "

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1. Peal, L., and Karp, M., A New Surface Anesthetic Agent: Tronothane. Anesthesiology, 15:637, November, 1954.

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Social News

Reported by K. Borthwick-Leslie, M.D.

December 22, 1955, St. Boniface Hospital at a staff meeting, honored a number of pioneers of the Medical Staff who for thirty years faithfully and conscientiously served the hospital. The following members were presented with a desk clock and calender along with a certificate: Dr. K. J. Backman, Dr. M. MacKay, Dr. L. Collin, Dr. J. P. Howden, Dr. J. Prendergast, Dr. W. F. Abbott, Dr. C. Clare, Dr. J. D. Adamson and Dr. F. D. McKenty.

♦

Speaking of pioneers, Dr. Murray Campbell (Thanks) has drawn to my attention the very interesting honor bestowed on his uncle, Dr. Robers Campbell, graduate of Manitoba Medical College, 1893, who received an honorary degree, (LL.D.) at the University of North Dakota. Dr. Campbell recently retired after sixty years in in practice in Grand Forks. Throughout this period he had been active in many civic affairs in the community, and a few months ago established a Foundation Fund at the University of North Dakota, of $160,000. Dr. Robert is a brother of Dr. A. M. Campbell in Winnipeg.

♦

At long last the University of Manitoba has set up a Surgical Research Laboratory under the supervision of Dr. Colin Ferguson, chairman of the Department of Surgery. The laboratory is at present being built in the east wing of the Medical Buildings. When completed it will greatly expand research facilities for staff members. Welcome contributions have been received from Hoffman La Roche, Wm. Warner Co., The Winnipeg Supply and Fuel Co., Bird Construction Co., Picker X-Ray Engineering Ltd., T. Eaton Co. Ltd., K. A. Powell, R. J. Gourley, J. K. May and A. H. Warner.

♦

Quote: Viennese born Richard Neutra, internationally famous architect, who is now in opposition to our Psychiatrists:

"Remember the fatigue phenomenon". You get tired of your one-toned car, your government, your wife. You know "shock" effects, you open the window wide, suddenly it's June—in Winnipeg.

Boy, oh boy, open the window right now and the shock is there alright—28° below—that's June for you, Mr. Neutra . . . Also I'm very curious as to what the architects have planned for that "'tired of your wife" theory, probably unique spacemaster shutters to replace the old fashioned "bay-window".

♦

Dr. D. F. Simpson, M.D., '53, formerly of Wilkie, Sask, is now practicing at Edmore, North Dakota.

Members of our Doctor Committee, re M.M.S. fees: Dr. Paul L'Heureux, D. J. Hastings, A. T. Gowron, M. K. Kiernan, L. R. Rabson, Ruvin Lyons, chaperoned by Doctors MacFarland and Thorlakson are, I believe, deep in thought and argument in Chicago, gleaning pearls of wisdam from the International Meeting in Chicago.

With fingers crossed we hope they return with some sane and sensible solution to our many problems. That extra billing has me worried.

♦

Dr. A. A. (Sandy) Campbell with his wife (nee Pat Rutherford) and son Peter will return to Winnipeg in April. Sandy and Pat have spent the last eight months in Edinburgh and northern England. Sandy will be in practice with his father, Dr. A. M. Campbell and brother Murray. Welcome home.

♦

Welcome also to the Medical Arts, to Dr. Christina Curran, who is now located in 120. Tough luck, Chris, you just made the fringe of the "Coral Corridor."

♦

Announcing the new arrival of the "St. James Medical and Dental Centre", 2031 Portage Ave., telephone 6-4851.

Drs. Henderson, O'Toole and Derbach are now occupying their new deluxe quarters, with Dr. Shorthill looking after the Dental Dept. Good luck to all.

♦

Dr. S. Stefan Bjornson, M.D., '46, with Mrs. Bjornson and three children are residing in Wilmington, Delaware. Dr. Bjornson has accepted an appointment as medical examiner for the State of Delaware.

♦

Belated announcement in the Bulletin of the marriage in New York City, December 3, 1955, of Miss Beverley Hader of Miami Beach to Dr. L. Mutchnik. The honeymoon was spent in Jamaica. Dr. Mutchnik is associated with Dr. Max Rady, in Winnipeg.

♦

Dr. and Mrs. A. M. Homik announce the birth of John Christopher David, January 16, 1956.

♦

Dr. and Mrs. Victor Rosenfield happily announce the appearance of Lorne Murray, brother for Mark, Barbara and Stephen.

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1955 Jan. 1 to Jan. 28,'56	1955 Dec. 4 to Dec. 31,'55	1954 Jan. 1 to Jan. 29,'55	1954 Nov. 28 to Dec. 25,'54
Anterior Poliomyelitis	0	2	0	0
Chickenpox	84	125	157	204
Diphtheria	0	4	1	0
Diarrhoea and Enteritis, under 1 year	4	32	2	5
Diphtheria Carriers	0	0	0	0
Dysentery—Amoebic	0	0	0	0
Dysentery—Bacillary	1	2	1	0
Erysipelas	3	1	0	0
Encephalitis	0	1	0	0
Influenza	5	12	6	6
Measles	138	199	251	140
Measles—German	5	4	4	0
Meningococcal Meningitis	1	5	2	3
Mumps	112	110	114	111
Ophthalmia Neonatorum	0	0	0	0
Puerperal Fever	0	0	0	0
Scarlet Fever	18	13	23	31
Septic Sore Throat	0	0	0	2
Smallpox	0	0	0	0
Tetanus	0	0	0	0
Trachoma	0	0	0	0
Tuberculosis	15	55	16	37
Typhoid Fever	0	0	0	0
Typhoid Paratyphoid	1	0	0	0
Typhoid Carriers	0	0	0	0
Undulant Fever	0	0	0	0
Whooping Cough	9	18	63	115
Gonorrhoea	99	104	80	91
Syphilis	3	6	4	5

Four-week Period January 1st to January 28th, 1956

DISEASES (White Cases Only) *Approximate population.	*828,000 Manitoba	*861,000 Saskatchewan	*2,825,000 Ontario	*2,952,000 Minnesota
Anterior Poliomyelitis	....	....	1	1
Chickenpox	84	8	2396	....
Diarrhoea and Enteritis, under 1 Yr.	4	27	....	....
Diphtheria	....	1	....	7
Diphtheria Carriers	....	....	....	....
Dysentery—Amoebic	....	....	....	....
Dysentery—Bacillary	1	....	11	5
Encephalitis Infectious	....	....	1	....
Erysipelas	3	1	5	....
Influenza	5	....	7	3
Jaundice Infectious	12	109	76	61
Measles	138	49	4003	24
German Measles	5	1	742	....
Meningitis Meningococcus	1	3	8	3
Mumps	112	5	2191	....
Ophthal. Neonat.	....	....	....	....
Puerperal Fever	....	....	....	....
Scarlet Fever	18	5	478	90
Septic Sore Throat	....	52	2	68
Smallpox	....	....	....	....
Tetanus	....	....	....	....
Trachoma	....	....	....	....
Tuberculosis	15	20	73	7
Typhoid Fever	....	....	4	7
Typh. Para Typhoid	1	....	....	....
Typhoid Carriers	....	....	....	....
Undulant Fever	....	....	3	5
Whooping Cough	9	31	201	22
Gonorrhoea	99	....	117*	....
Syphilis	3	....	14*	....

*Statistics for three weeks only

DEATHS FROM REPORTABLE DISEASES
January, 1956

Urban—Cancer, 36; Pneumonia Lobar (490) 7; Pneumonia (other forms), 17; Syphilis, 1; Tuberculosis, 1; Diarrhoea and Enteritis, 3; Septicaemia and Pyaemia, 2. Other deaths under 1 year, 16. Other deaths over 1 year, 165. Stillbirths, 7. Total, 255.

Rural — Cancer, 23; Influenza, 2; Measles, 2; Pneumonia Lobar (490), 2; Pneumonia (other forms), 4; Tuberculosis 1; Undulant Fever, 1. Other deaths under 1 year, 23. Other deaths over 1 year, 126. Stillbirths, 3. Total, 187.

Indians—Pneumonia (other forms), 2. Other deaths over 1 year, 3. Other deaths under 1 year, 3. Stillbirths, 0. Total, 8.

♦

Poliomyelitis—actually one case did occur in mid January but was not officially reported until after the four week period had ended. This was a nine year old boy who developed Polio with slight paralysis on January 16th, 1956. He is now almost totally recovered. His five year old sister had become a case with moderate paralysis on December 29th, 1955. The boy had two doses of Salk Vaccine in April and May 1955—the sister had no vaccine. Ths case proves nothing excepting the fact that two doses of vaccine do not **completely** protect 100% of the people. If this boy had not received vaccine he **might** have had marked paralysis and even died or he **might** have been no worse. The point is that he has had Poliomyelitis and is practically not harmed by it. Only time and larger statistics will give us a true evaluation of the Salk Vaccine.

Chickenpox, **Measles** and **Mumps** are quite prevalent.

Other than the above "all is reasonably well."

Cancer Reporting in Manitoba

The Regulations under the Public Health Act state that: "Cancer shall be deemed to be a notifiable disease under the Act". From time to time the report form supplied by the Department of Health and Public Welfare has been revised so as to obtain more accurate and valuable information. Consequently there are several types of forms in existence. The most recent revision is of January 1956 (at the request of the Cancer Relief and Research Institute). A supply of these new forms has been distributed to each hospital in the province as most reports are sent in from the hospitals.

We would ask you physicians at this time to **destroy** any of the **old cancer report forms** you have in your office and if you see cancer cases in your office which will not be reported through hospitals please telephone or write the Department asking for a supply of the new forms and return envelopes. Address requests to the Department at 320 Sherbrook Street, Winnipeg 2, or telephone SUnset 3-7131 and ask for Preventive Medical Services.

M. B.

Salk Poliomyelitis Vaccine 1956

The Department of Health and Public Welfare of the Province of Manitoba expected to have a quantity of this vaccine available early in March to continue the program of vaccination against poliomyelitis.

Our latest information is that we cannot expect sufficient vaccine to start our program before late April or early May. We hope at that time to have sufficient vaccine available to give the third dose to the children who received two doses in 1955, and in addition, at least one dose to all remaining children in Public Schools throughout the province, up to and including Grade VIII. Second doses will be given to this latter group when more vaccine is available, and third doses at a still later date.

Book Review

Diagnosis of Congenital Heart Disease by Sven Kjellberg, Edgar Mannheimer, Ulf Rudhe, and Bengt Jonsson, the Cardiac Team at the Paediatric Clinic, Karolinska Sjukhuset, Stockholm, Sweden. 641 pages; 1845 illustrations on 618 figures; 36 tables. $22.00. Published by the Year Book Publishers, inc., Chicago, Ill.

The publishers announce this book as one "destined to be a classic in this field". Laudatory as the epithet "classic" may be, it has often the effect of scaring away the potential reader, thus consigning the book to a dust gathering future on a library shelf. It would be a pity, indeed, if this fate befell the masterful work of the Karolinska Sjukhuset Cardiac Team, for the book is very readable, its heavy content lightened by its lucidity of style and artistic quality.

This book is divided into two parts. The first part deals with embryology, anatomy, physiology and diagnostic procedures, the latter including electrocardiography, calibrated phonocardiography, blood-gas analysis, electrokymography and roentgenologic examination. The second part summarizes the experience of the investigating team with 342 cases of congenital malformations. Although dealing on the whole with material that came under their personal observation, the writers have included an extensive bibliography with up to date references.

The most striking feature of this book is the wealth of its pictorial content. It boasts of 1845 illustrations, which include diagrams of intracardiac circulation, photographs of anatomical specimens, electrocardiograms, phonocardiograms and roentgenograms. The latter, numbering 1404, include what may be regarded as a most comprehensive and up to date atlas of angiocardiography.

The book is neither an atlas, nor a textbook, nor a reference book in a narrow sense of the word, but rather a thorough and critical study of extensive case material in a new and widely expanding field of medicine. It is not, however, without faults and shortcomings, the most regrettable being the absence of a summary and conclusion at the end of each chapter, which would help to tie the loose ends together. It is, indeed, somewhat disappointing to find a long chapter on pulmonary stenosis end in a relatively unimportant observation on a postoperative aneurysm of the ventricle. Similarly, the last paragraph of the book, consisting of a comment on the width of the vessel in "idiopathic" dilatation of the pulmonary artery, comes as an anti-climax.

Another adverse criticism may be directed at the classification adopted by the writers. One may agree with them that "it is not possible to make a consistent classification of congenital heart disease on the basis of presence of shunts and their direction", yet find their alternative, the anatomic classification, which is more in the nature of a tabulation, even less satisfying to the understanding and memory.

Let not these critical remarks, however, detract from the value of the book, for its imperfections are redeemed by its overall excellence. It is recommended reading for all who are interested in congenital heart disease.

S. V

Senior Interne Required

Applications for the position of Senior Interne in Internal Medicine at Deer Lodge (D.V.A.) Hospital for the year beginning July 1, 1956, are invited. Deer Lodge Hospital is approved for advanced graduate training by the Royal College of Physicians and Surgeons of Canada. Apply to the Superintendent.

Pathologist Required

"Pathology Residencies and Fellowships available commencing June 1, 1956 at the Winnipeg General Hospital, approved for training by The Royal College of Physicians and Surgeons and for American Boards. Apply to: The Director, Department of Pathology, Winnipeg General Hospital, Winnipeg 3, Man.

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